# The American Journal of Surgery

EDITED BY

*Thurston Scott Welton*, M.D., F.A.C.S.


SPECIAL MONOGRAPH

## Diagnosis and Treatment of Various Types of Diaphragmatic Hernia

By STUART W. HARRINGTON


*November* 1940

THE AMERICAN JOURNAL OF SURGERY, Inc.

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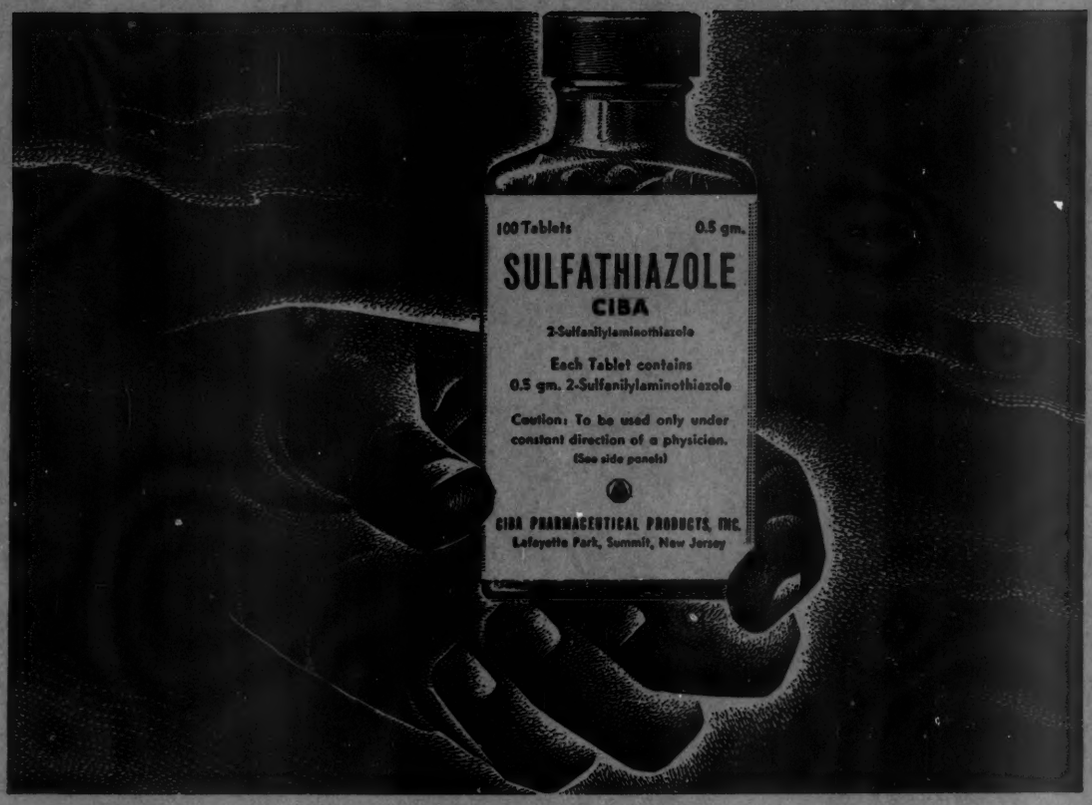


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# *The Rationale of Suture Use*

BRIEF DISCUSSIONS BASED ON REPORTS OF CURRENT RESEARCH AND CLINICAL OBSERVATION

## What Tissues Should Be Sutured?

THE usual concept of suturing is to approximate all layers, to collapse all dead space, but this should be further qualified regardless of the type of suture material employed.

Tissues to be sutured are:

- Those which tolerate sutures well.
- Those which provide the most immediate strength for the wound.
- Those which will not separate again after suturing.
- Those which protect the deeper structures from further bacterial contamination.
- Those which provide mechanical advantages other than strength.
- Those which can be approximated without tension.
- Those which are free from gross bacterial contamination.

Fasciae, ligaments, capsules of organs and the sheaths of muscles provide the greatest immediate strength for the sutured wound, are least likely to release sutures later, and tolerate sutures well. On the other hand, fat, muscle substance, and the pulp of parenchymatous organs have practically no suture strength, tolerate sutures poorly, necrotize easily, and tend to separate again after suturing.

Suturing of skin and mucous membranes protects the deeper structures from bacterial contamination, but the skin does not tolerate perforation with absorbable sutures, and mucous membranes tolerate only those which are exceedingly fine. Fine absorbable subcuticular or sub-mucosal sutures may be used successfully to approximate these structures.

Peritoneum is sutured to prevent wedging of omentum or intestines, and thus prevent a disruption of the wound or a post-operative hernia. This principle is furthered by a use of a continuous stitch.

In general, tissues are under too much tension if the cut edges cannot be approximated by a single stitch of size 0 without the suture breaking. Plastic release incisions should be employed rather than to attempt to approximate the edges with sutures of larger size.

Recent war experience has emphasized again that bacteria begin to invade traumatic wounds seven hours after infliction. Wounds in this condition should not be sutured.

*Bibliography or answers to specific questions on this subject will be sent on request.* DAVIS & GECK, INC., *Brooklyn, N. Y.*

# D&G Sutures

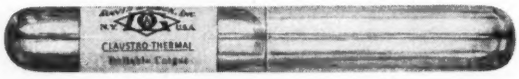
**NOTE: In agreement with the new U. S. P. nomenclature, catgut products formerly known as Plain, 10-day, 20-day, and 40-day, are now designated as**

**Plain – type A** — **Medium chromic – type C**

**Mild chromic – type B** — **Extra chromic – type D**

THERE HAS BEEN NO CHANGE IN THE PRODUCTS THEMSELVES

## Claustro-thermal Catgut




POSSESSES all the qualities essential to proper function and is adaptable to all conditions and technics. The tubes may be boiled or autoclaved for asepticization of their outer surface or treated germicidally.

This catgut offers the additional safety factor of CLAUSTRO-THERMAL★ sterilization—an exclusive D&G process wherein heat, at temperatures lethal to the most resistant spores and organisms, is applied AFTER the suture is hermetically sealed in its glass tube and thus fully protected.

| NO. | | LENGTH *in excess of* | SIZES |
|---|---|---|---|
| 1205 | Plain—type A | 54″ | 5-0 to 4 |
| 1225 | Mild chromic—type B | 54″ | 4-0 to 4 |
| 1245 | Medium " —type C | 54″ | 4-0 to 4 |
| 1285 | Extra " —type D | 54″ | 4-0 to 4 |

Package of 12 tubes of a kind $3.60

## Thermo-flex Catgut

FLEXIBILITY is the feature of this catgut. Ready for use as it comes from the tubes, it requires no special preparation.

Though of the THERMO-FLEX,★ *non-boilable*, type, there is no compromise in sterility. It is sterilized by HEAT at temperatures exceeding the most rigid bacteriologic requirements.

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|---|---|---|---|
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| 1425 | Mild chromic—type B | 54″ | 4-0 to 4 |
| 1445 | Medium " —type C | 54″ | 4-0 to 4 |
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## Kal-dermic Sutures

KAL-DERMIC★ represents the culmination of eight years' research to perfect a skin and tension suture combining the best features, while eliminating the disadvantages of silkworm gut, horsehair, silk and several synthetic products variously named.

Kal-dermic is completely stable. It is highly resistant to tissue fluids and germicides; its color is permanent under all conditions; and the strand may be repeatedly boiled or autoclaved without impairment.

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| NO. | NEEDLE | LENGTH | SIZES | PER BOX |
|---|---|---|---|---|
| 550 | None | 2 x 60″ | 4-0 to 0 | $3.60 |
| 953 | ⅜-circle | 20″ | 000, 00, 0 | 3.00 |
| 954 | Half-curved | 20″ | 000, 00, 0 | 3.00 |
| 852 | None | 40″ | 8-0, 6-0, 4-0 to 0 | 1.80 |

**On reels, unsterilized**
(Ten reels to a box)

| | | | NET, PER DOZ. BOXES |
|---|---|---|---|
| 50 | None | 190″ | 0000 $10.50 |
| | | | 000 11.64 |
| | | | 00 12.60 |
| | | | 0 14.40 |

### KAL-DERMIC TENSION SUTURES

**In tubes, sterilized**
(Twelve tubes to a box)

| | | | | PER BOX |
|---|---|---|---|---|
| 555 | None | 60″ | 1 to 4 | $3.60 |
| 855 | None | 20″ | 1, 2, 3 | 1.80 |

**On reels, unsterilized**
(Ten reels to a box)

| | | | NET, PER DOZ. BOXES |
|---|---|---|---|
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| | | | 2 20.16 |
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| NO. | NEEDLE | LENGTH *in excess of* | SIZES | DOZEN |
|---|---|---|---|---|
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| 50344 | Half-circle | 28″ | 5-0 | 4.20 |
| 50245 | None | 54″ | 5-0 | 3.60 |
| 40341 | Straight | 28″ | 4-0 | 3.60 |
| 40344 | Half-circle | 28″ | 4-0 | 4.20 |
| 40245 | None | 54″ | 4-0 | 3.60 |

THERMO-FLEX (*non-boilable*)

| | | | | |
|---|---|---|---|---|
| 50541 | Straight | 28″ | 5-0 | $3.60 |
| 50544 | Half-circle | 28″ | 5-0 | 4.20 |
| 50445 | None | 54″ | 5-0 | 3.60 |
| 40541 | Straight | 28″ | 4-0 | 3.60 |
| 40544 | Half-circle | 28″ | 4-0 | 4.20 |
| 40445 | None | 54″ | 4-0 | 3.60 |

Packages of 12 tubes of a kind and size

## Anacap Silk

NEW principles of fabrication provide ANACAP★ silk with unprecedented strength, knot security, and absolute non-capillarity.

| NO. | LENGTH | SIZES | DOZEN |
|---|---|---|---|
| A40 | 40″ | 8-0, 6-0, 4-0 to 4 | $1.80 |
| A120 | 2 x 60″ | 8-0, 6-0, 4-0 to 4 | 3.60 |

Packages of 12 tubes of a kind and size

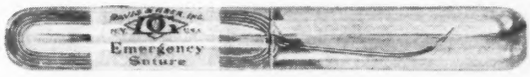
## Unabsorbable Sutures

| NO. | MATERIAL | LENGTH | SIZES |
|---|---|---|---|
| 350 | Celluloid-linen | 60″ | 000, 00, 0 |
| 360 | Horsehair | 6 x 28″ | 00 |
| 390 | White silkworm gut | 6 x 14″ | 00, 0, 1 |
| 400 | Black silkworm gut | 6 x 14″ | 00, 0, 1 |
| 450 | White twisted silk | 60″ | 000 to 3 |
| 460 | Black twisted silk | 60″ | 000 to 2 |
| 480 | White braided silk | 60″ | 00, 0, 2, 4 |

Packages of 12 tubes of a kind $3.60

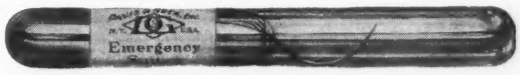
## Emergency Kit Sutures

THREADED on half-curved or 3/8-circle eyed needles with cutting edges for skin, muscle, or tendon. Heat sterilized in glass tubes which may be either boiled or submersed in any active germicidal solution to asepticize their outer surface.




WITH HALF-CURVED NEEDLES

| NO. | MATERIAL | LENGTH *in excess of* | SIZES |
|---|---|---|---|
| 904 | Plain—type A catgut | 18″ | 00 to 3 |
| 914 | Mild chromic—type B | 18″ | 00 to 3 |
| 924 | Medium chromic—type C | 18″ | 00 to 3 |
| 954 | Kal-dermic | 20″ | 000, 00, 0 |
| 964 | Horsehair | 2 x 28″ | 00 |
| 974 | White silkworm gut | 2 x 14″ | 00, 0 |
| 984 | White twisted silk | 20″ | 000, 0, 2 |
| 986 | Anacap silk | 20″ | 000, 0, 2 |
| 900 | Assorted: Catgut, Silk, and Kal-dermic | | |




WITH 3/8-CIRCLE NEEDLES

| | | | |
|---|---|---|---|
| 903 | Plain—type A catgut | 18″ | 00 to 2 |
| 923 | Medium chromic—type C | 18″ | 00 to 2 |
| 953 | Kal-dermic | 20″ | 000, 00, 0 |
| 963 | Horsehair | 2 x 28″ | 00 |
| 973 | White silkworm gut | 2 x 14″ | 0 |
| 983 | White twisted silk | 20″ | 000, 0, 2 |
| 985 | Anacap silk | 20″ | 000, 0, 2 |
| 930 | Assorted: Catgut, Silk, and Kal-dermic | | |

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## Other D & G Sutures

IN ADDITION to the foregoing a wide variety of suture-and-needle combinations is available for intestinal, thyroid, tonsil, eye, harelip, cleft palate, plastic, nerve, artery, obstetrical, circumcision, ureteral, renal and dental surgery. A complete list of sizes, lengths, needle combinations, etc. will be supplied on request.

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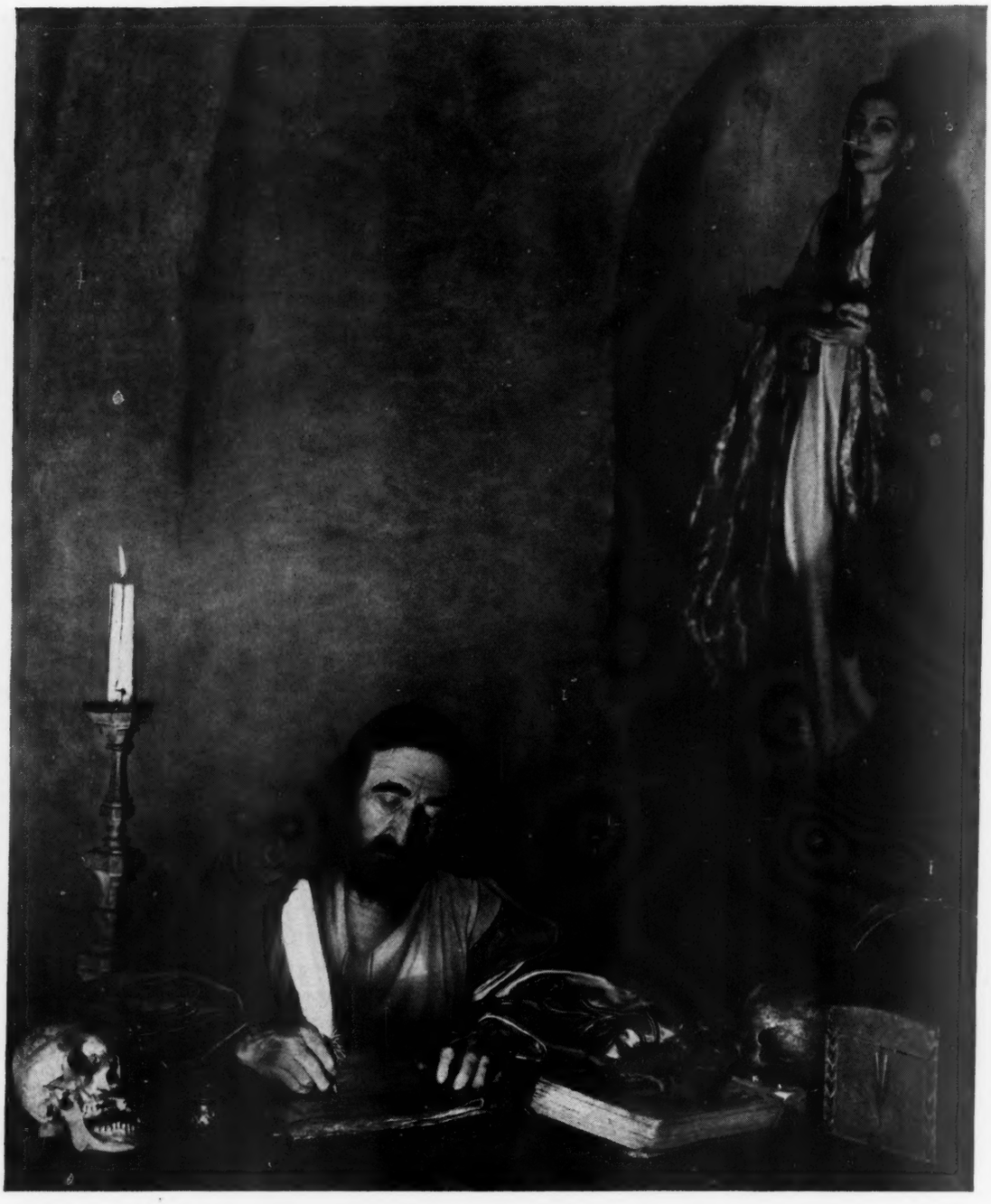

BRUNO OF CALABRIA whose *Chirurgica Magna* (1252) is notable as the first work to cite both Greek and Arabian writers, also compiled a *Chirurgica Minor* in 23 chapters. In the treatment of large wounds he advises careful suturing with silk or linen, one end of the wound to be left open and sutures to remain for eight days. For abdominal wounds he suggests suturing the peritoneum to the abdominal musculature and emphasizes the advantages of dry healing in all cases.

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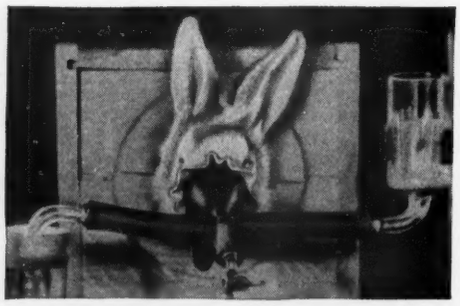

Part of experimental set-up for determination of respiratory volume.

*Trade Mark Reg. U.S. Pat. Off. Word "Coramine" identifies the product as the diethyl amide of nicotinic acid of Ciba's manufacture.

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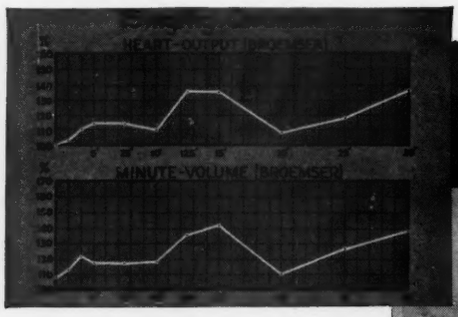


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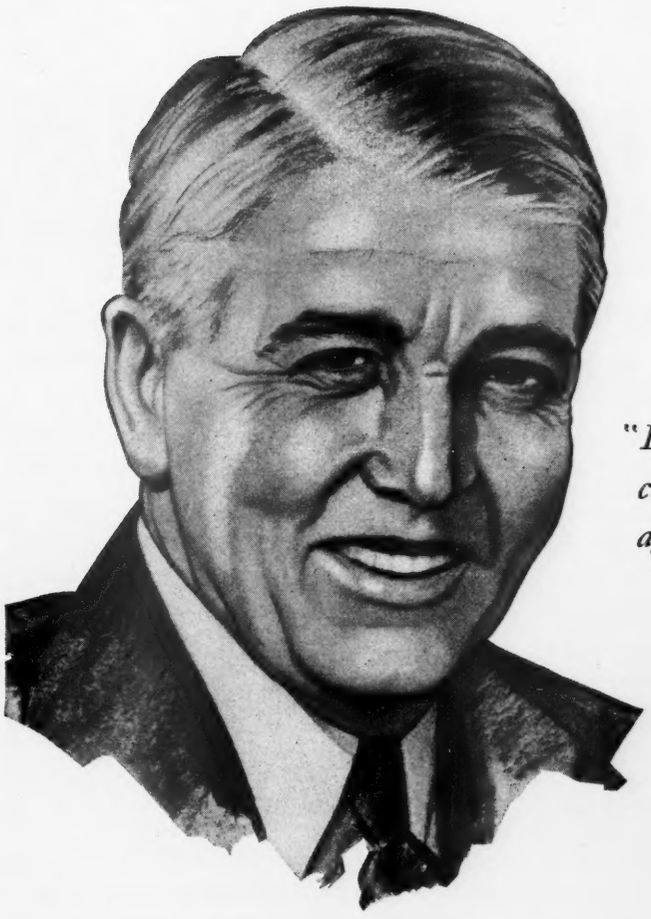

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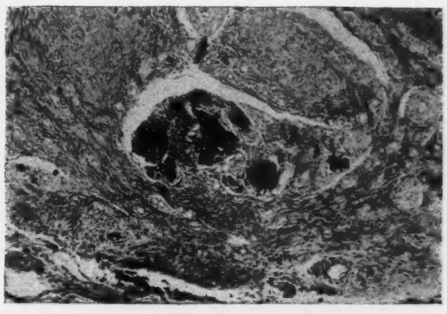
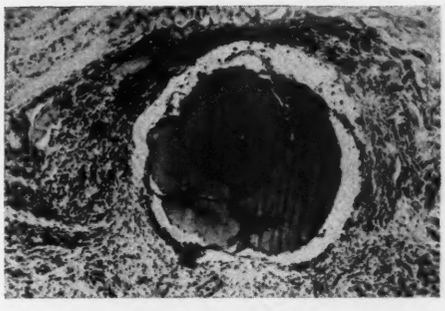


*An improperly chromicized strand is almost completely absorbed.*



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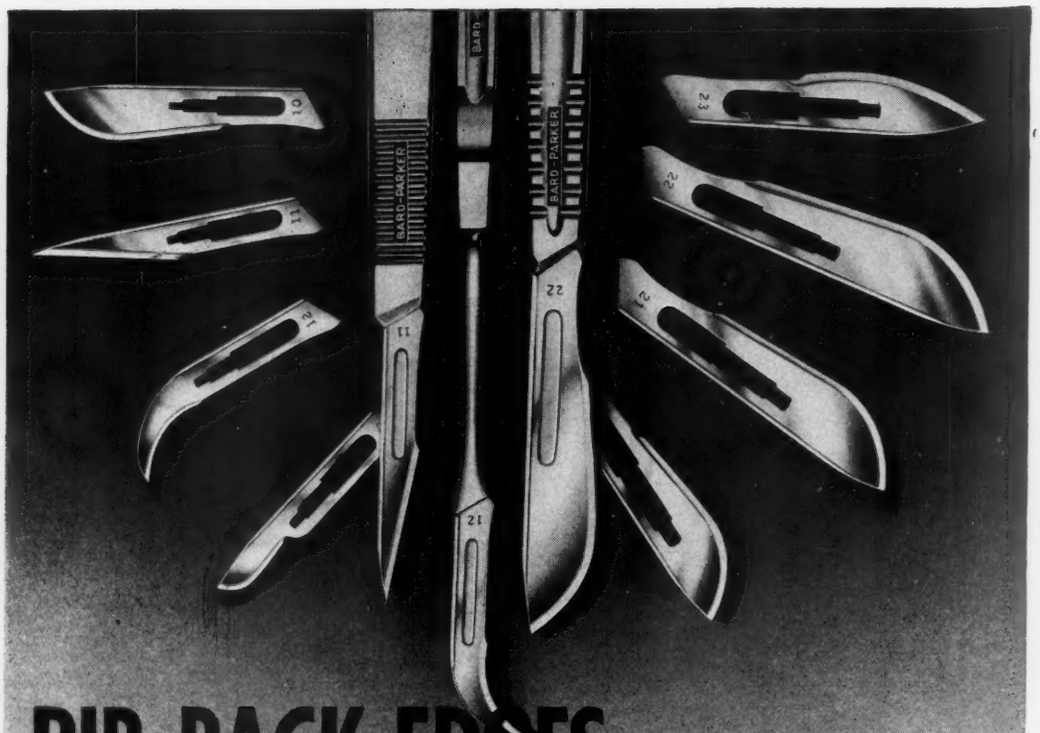


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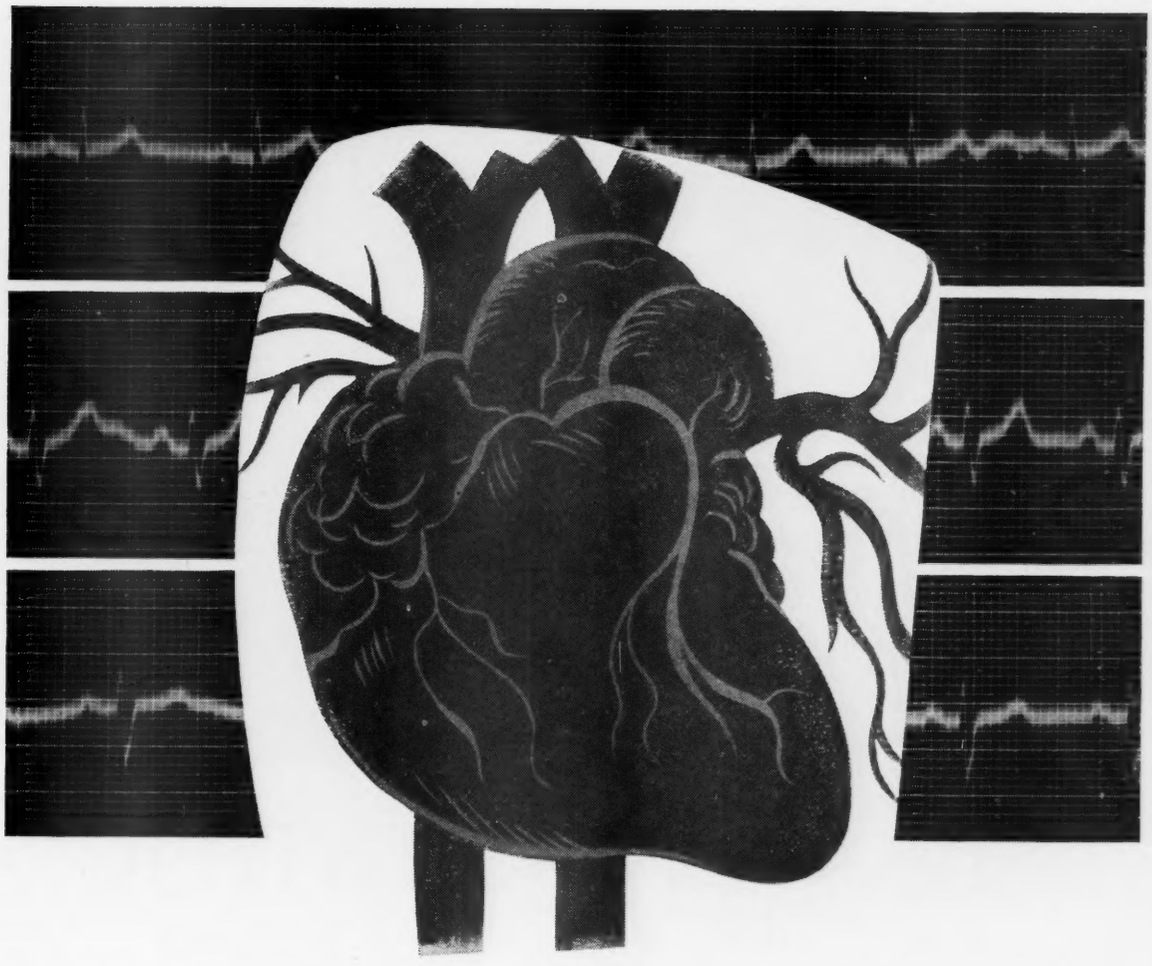

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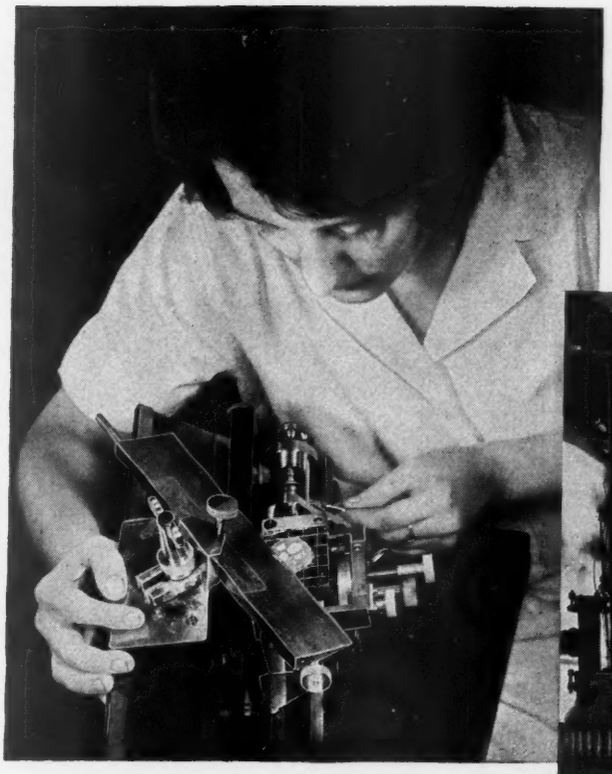

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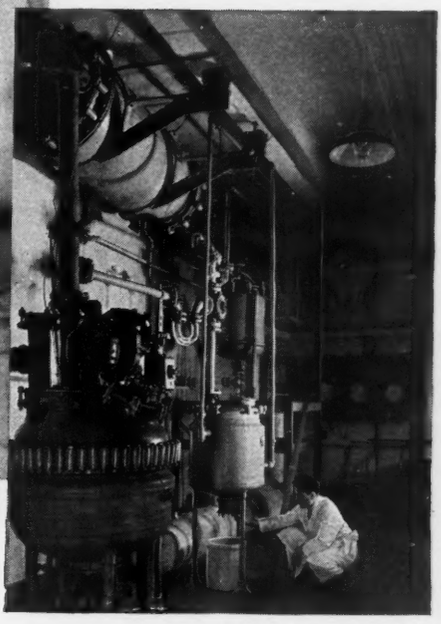

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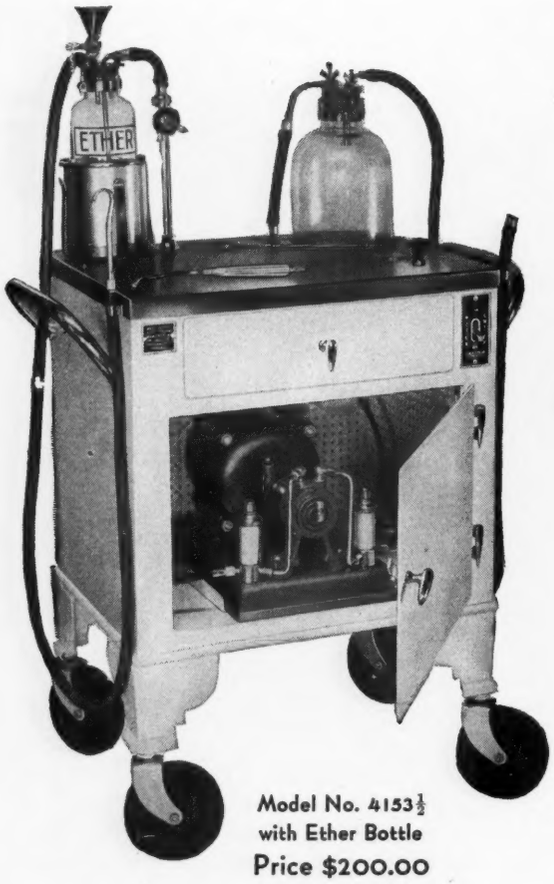


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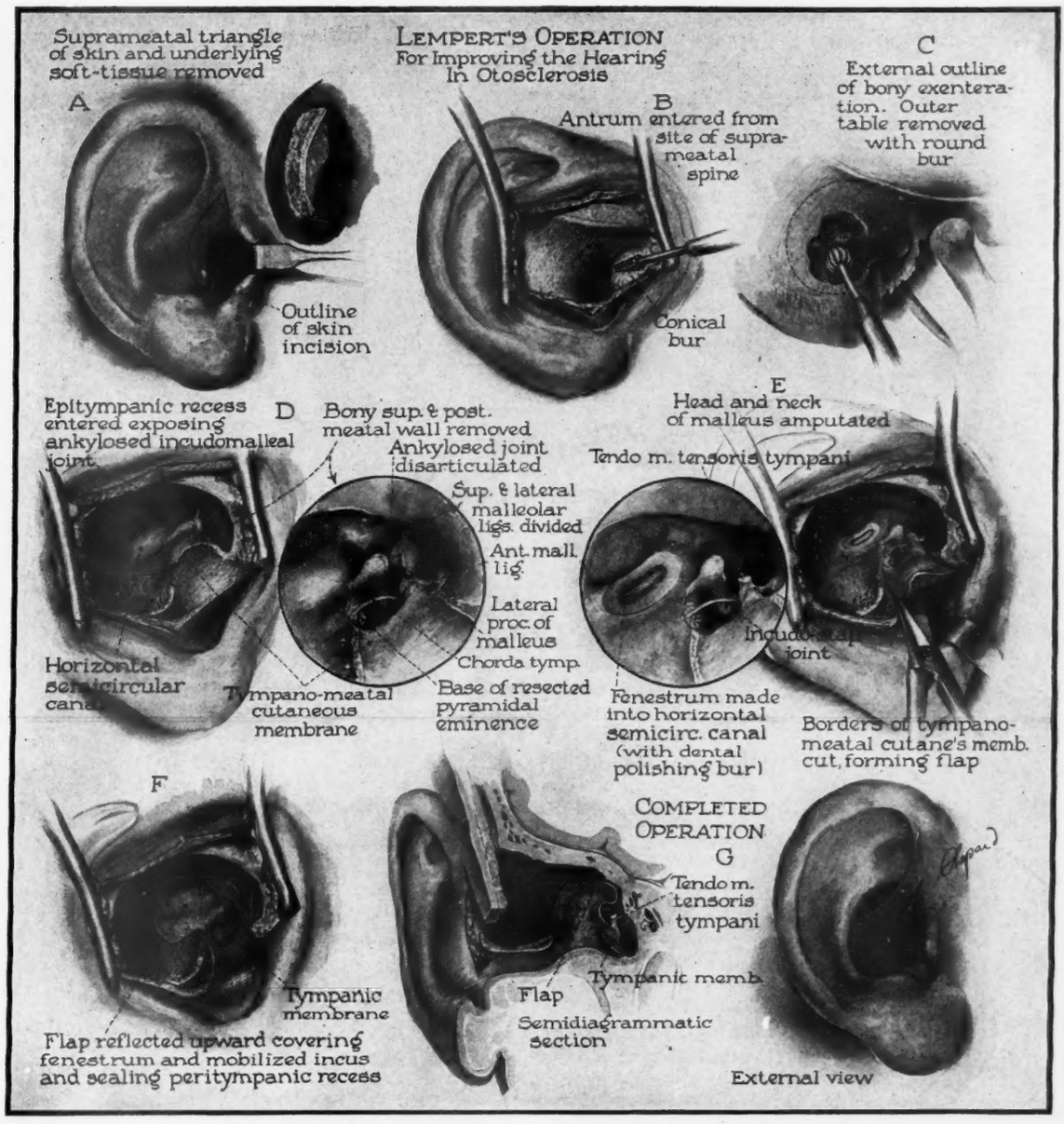


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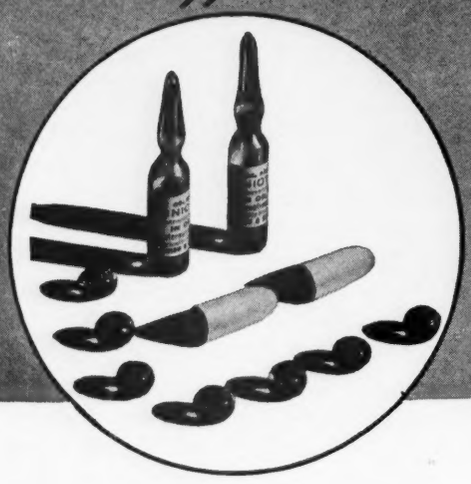

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[1] Sevringhaus, E. L., and Evans, J. S.: *Am. J. M. Sc.* 178:638, Nov. 1929.
[2] Novak, Emil: *Surg. Gynec. & Obst.* 70: 124, Jan. 1940.
[3] Schneider, P. F.: *Am. J. Obs. & Gyn.* 37:861, May 1939.

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Amniotin — A SQUIBB PREPARATION OF ESTROGENIC SUBSTANCES OBTAINED FROM THE URINE OF PREGNANT MARES

NEW SERIES, VOL. L NOVEMBER, 1940 NO. 2

The American Journal of Surgery

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# EDITORIAL

## MONOGRAPHS

IN an editorial entitled "Use and Abuse of Advertising Brochures," which appeared in the Journal of the American Medical Association (October 5, 1940), the last paragraph was of special interest to us. In part it read, "The whole field of medical publication is undergoing an obvious transformation. The speed of the times would seem to demand more in the nature of monographic publications dealing with a single subject and less of extensive textbooks which tend to become obsolete, at least in portions of their text, in a short time. Development of this trend for the monograph in the field of unbiased literary production would be useful to the medical profession . . . "

The American Journal of Surgery was a pioneer in presenting to its readers short monographs by outstanding men in their fields of activity. During the past two years we have published the following monographs:

Arthur E. Hertzler—"Technique of Thyroidectomy," November 1938.

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Arthur Goetsch—"The Diagnosis and Treatment of Hyperthyroidism and Associated Conditions," September 1940.

George J. Heuer and William DeWitt Andrus—"The Surgery of Mediastinal Tumors," October 1940.

Stuart W. Harrington—"Diagnosis and Treatment of Various Types of Diaphragmatic Hernia," which appears in this current issue.

We agree that the monograph is a valuable addition to surgical literature. It contains in a nut shell what a reader is looking for without his being obliged to wade through numerous articles on different phases of a given subject. And we have also noticed that other surgical publications have featured monographs since and long after we started the practice.

Then, too, we are of the opinion that the written-to-order Special Numbers, edited by surgeons of outstanding reputation, are of great value. In cases in which a monograph would not cover the topic adequately, a series of articles by recognized authorities have been written on subjects more or less related. These articles, appearing in one issue, have proved popular and our readers have asked that we continue this practice. During the past two years we have published the following symposia:

"Traumatic and Industrial Surgery," edited by Dr. Allen D. Lazenby, December 1938.

"Reconstructive and Plastic Surgery," edited by Dr. Fred H. Albee, February 1939.

"Surgery of the Extremities," edited by Dr. W. Wayne Babcock, April 1939.

"Large Intestine," edited by Dr. Charles Gordon Heyd, October 1939.

"Emergency Surgery," edited by Dr. Roy D. McClure, December 1939.

"Obstetrics and Gynecology," edited by Dr. Fred L. Adair and Dr. Emil Novak, April 1940.

In our forthcoming issue for December we have a very important and practical Special Number on Office and Minor Surgery arranged and edited by the author.

The practice of publishing monographs and special numbers will be continued by this Journal. We are always searching for topics we hope will prove both instructive and interesting. Therefore, suggestions from our readers are always welcome. Our main ambition is to publish a Journal that will be valuable, readable and to your liking.

T.S.W.

# ORIGINAL ARTICLES

## TRIGEMINAL NEURALGIA WITH DEMONSTRABLE GROSS CAUSATIVE LESIONS*

### REPORT OF FIVE CASES

RUPERT B. RANEY, M.D. AND AIDAN A. RANEY, M.D.

LOS ANGELES, CALIFORNIA

FREQUENTLY pathologic causes of major trigeminal neuralgia are not clearly demonstrable. This statement pertains to the classic type of the disease described by Fothergill (1773),[1] i.e., the disease which is characterized by: (1) severe paroxysmal attacks of darting pain confined to the distribution of one or more branches of the trigeminal nerve; (2) attacks occurring spontaneously or precipitated by stimulation of trigger zones; (3) the absence of neurologic findings.

Many other types of facial neuralgia that do not conform to Fothergill's description have generally been called atypical. Unless the disease is characteristic of the classic type, relief by surgical treatment has usually been reported as unsatisfactory. The so-called atypical syndromes have even been considered by some authors to be contraindications to surgical treatment; while this generalization is justified in some cases, there are notable exceptions.

Any further classification of facial neuralgia, aside from the classic and atypical types, is almost impossible. However, of the atypical cases two general groups can be recognized, viz., those which can be expected to respond to surgical treatment, and those which cannot be expected to respond. In the former group the pain may vary considerably in character, but it follows the anatomic distribution of one or more branches of the trigeminal nerve, and neurologic findings are usually present. In the atypical cases which probably will not respond to surgery, these conditions are generally not present.

The first authentic case of major trigeminal neuralgia (classic tic douloureux) recognizable as such, was that of Dr. Johannes Laurentius Bausch, reported by Johannes Michael Fehr and Elias Schmidt (1771), five years after the death of the patient.[2] The first accurate account, however, was by Fothergill. According to the record of his cases, objective neurologic findings either were not recorded or were absent.

At Spiller's suggestion, Frazier (1901)[3] established a safe operation for the classic type of the disease, namely, posterior root section; this gave uniform and permanent relief. More recently Sjöqvist (1938)[4] made an extensive fiber analysis of the trigeminal tract and corroborated and added to the earlier findings of Adrian and Zotterman (1926),[5] Gasser and Erlanger (1927),[6] and others, showing that the pain and temperature fibers segregate themselves in the pons and descend downward in the trigeminal tract through the medulla. He further pointed out that section of the trigeminal tract in the medulla at the level of the inferior olive gave uniform relief from major trigeminal neuralgia. Moreover, the procedure allows exploration of the cerebellopontine angle for possible causes and gives anesthesia for only pain and temperature, while touch and pressure are preserved.

* From the Department of Surgery, University of Southern California School of Medicine and the Neurosurgical Service, Los Angeles County Hospital.

Numerous other reports have appeared in the literature with reference to treatment, e.g., alcohol injection, trichlorethylene, and more recently vitamin therapy. Of these, alcohol injection can reasonably be expected to give temporary relief; other forms of treatment, however, have generally been unsatisfactory. Thus, it becomes apparent that permanent relief in the classic type of case can be expected only by section of the sensory root of the trigeminal nerve, as originally advocated by Spiller and Frazier, or by tractotomy as recommended by Sjöqvist.

A considerable number of contributions regarding etiology have been made, but in comparatively few instances have actual causes been definitely established. Parker (1928)[7] reported four cases of major trigeminal neuralgia which developed during the course of multiple sclerosis; in one, post-mortem examination showed sclerotic plaques on the descending root and nucleus of the trigeminal nerve at the level of the inferior olive; other plaques involved the root of the nerve in the pons.

According to Dandy (1934)[8] vascular anomalies and neighboring neoplasms, such as tortuosity of the basilar artery, venous anomalies of the petrosal sinuses, acoustic neuroma, etc., have caused major trigeminal neuralgia by direct pressure on the root of the trigeminal nerve, relief being obtained by removal of the cause or radicular section. Trauma has been described as an etiologic factor by Alajouanine and Thurel (1937),[9] Colucci (1936),[10] Aubertine (1936),[11] and others. Hoff (1934)[12] reported a case that followed the administration of tuberculin for pulmonary tuberculosis. Harris (1935)[13] described four cases in which neuralgia developed after an attack of acute trigeminal neuritis; in his cases, there was a sudden onset of hemianesthesia of the face, followed by partial recovery of sensation and severe attacks of major trigeminal neuralgia. Hevia (1937)[14] reported tuberculosis as an etiologic factor.

Recently Lewy and Grant (1938)[15] reported focal softening located either in the lateral and medial nuclei of the optic thalamus, or in the thalamocortical tract. Furthermore, they observed that in 25 per cent of their patients the number of touch and pain points was reduced in the distribution of the trigeminal nerve (penesthesia and penalgesia), and that the preserved points showed a greater stimulus threshold than normal. In 32 per cent of the patients showing sensory changes, they reported that the changes extended to the corresponding half of the body, and that forty of the fifty patients studied showed pyramidal or extrapyramidal tract involvement.

Thus, it appears that major trigeminal neuralgia probably has a variety of etiologic factors. The so-called atypical syndromes, with which we are here concerned, differ principally from the classic syndrome in that pathologic causes and objective physical signs of organic disease can both be demonstrated. The discussion of a large group of other facial neuralgias having a nondescript character has been omitted since they require an entirely different management; if treated surgically, such patients are generally not benefited. In the cases here reported the character of the pain was somewhat variable, but its distribution was fairly typical of the classic type of the disorder, i.e., it followed the anatomic distribution of the trigeminal nerve. On the other hand, certain atypical features uniformly presented themselves, viz., etiologic factors were established, and objective neurologic signs of organic disease were present.

## REPORT OF CASES

CASE 1. *Head injury in a young man followed immediately by continuous left trigeminal pain; later attacks of major trigeminal neuralgia in the second and third divisions of the affected nerve. Objective findings of left primary optic atrophy, left temporal heminopia with loss of macular vision, and hypesthesia in all three divisions of the affected trigeminal nerve. Fractional section of the posterior root gave relief.*

The patient, a man of 35 years, was admitted to the hospital September 17, 1933, a few hours after he had sustained a head injury. When admitted, he was quite drowsy and very evidence of fracture. Spinal puncture gave a pressure of 120 mm. of water; no cells were found. The Wassermann reaction of both blood and spinal fluid was negative.

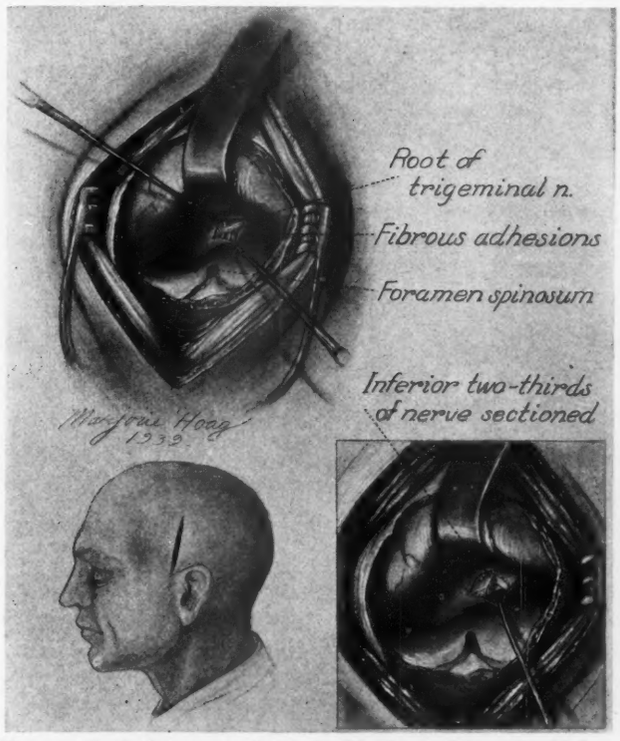


Fig. 1. Artist's conception of adhesions that existed between the dura propria and the posterior root of the trigeminal nerve, supposedly the result of trauma.

little history could be obtained. On the following day he was more alert, but did not know how long he had been unconscious or how he had been injured; other informants told us that he had been struck over the left eye with a shovel, and had been rendered unconscious for less than an hour.

Even on the day of injury while still drowsy he complained of continuous and severe pain in the left side of the face; he also complained of dizziness on change of position. Examination showed: (1) a bruise over the left forehead, (2) semistupor, (3) spontaneous rotary nystagmus, (4) loss of left direct pupillary reaction to light, but normal consensual and convergence reactions. On the following day the patient had regained consciousness, and had the additional complaint of loss of vision in the left eye. Fundus examination revealed nothing abnormal. The visual fields showed complete loss of vision in the left eye except for the inferior nasal quadrant; all macular vision was lost. Roentgenograms of the skull showed no

Ten days later the swelling over the forehead had disappeared; the dizziness had largely subsided, but he still complained bitterly of pain through all three divisions of the trigeminal nerve on the left side. A perimetric field study showed that vision had failed to make any improvement.

Five weeks after the accident the patient was discharged from the hospital, still complaining of pain. The left visual field still showed the same defect; there had been no return of macular vision, and there was beginning primary optic atrophy. After dismissal from the hospital he returned to the outpatient clinic at weekly intervals. The facial pain had not diminished; the visual fields had remained essentially the same, as had the pupillary reflexes.

One year after the accident there had been no improvement in his condition. In addition he was then suffering not only from constant pain but also from paroxysmal attacks of darting pain in the left maxillary and mandibu-

lar regions; there were no trigger zones present. Alcohol injection in the maxillary branch of the nerve was unsatisfactory. Partial relief, for four months, was obtained by injection of the mandibular branch, but then the pain returned with greater severity than ever.

Twenty-one months from the date of the accident (June 12, 1935) the patient was readmitted to the hospital because of severe paroxysmal attacks of darting pain in the second and third divisions of the nerve, as well as constant aching pain through all three divisions. Examination gave essentially the same findings as previously: (1) primary optic atrophy with loss of pupillary reactions to light in the affected eye; (2) hypesthesia in all three branches of the left trigeminal nerve.

Five days later (June 26, 1935) the ganglion and the posterior root of the left trigeminal nerve were explored under local anesthesia. The dura was found to be extremely adherent to the temporal bone, especially in the region of the petrous tip. When the dura propria was opened no spinal fluid escaped. The fibers of the nerve root were adherent to one another and also to the dura propria. The inferior two-thirds of the root were sectioned. (Fig. 1.) During the operation the patient continued to complain of pain, but after the section was performed he felt quite comfortable. A sensory check of the face revealed complete loss of sensation through the mandibular and maxillary branches of the nerve; sensation of cornea, upper eyelid, and forehead showed only the amount of impaired sensation that had been present since the accident. Since there had been no paroxysmal pain in the ophthalmic division of the nerve, the root was not completely sectioned and corneal sensation was preserved.

Twelve days after operation there had been no return of pain. The patient stated that he felt a dull drawing sensation about the forehead and upper eyelid, but that it was not painful. He was then discharged from the hospital with slight drainage from the wound which healed completely by the end of four weeks. He returned at monthly intervals for the following year. When he was last seen (July 6, 1939) four years after operation, there had been no return of pain and he had been working for two and one-half years; other physical findings were essentially the same.

*Comment.* In this patient trauma must have played an important rôle in the production of trigeminal neuralgia. Furthermore, the syndrome was atypical in several respects: (1) no trigger zones were present; (2) there was continuous dull pain between the sharp paroxysmal seizures; (3) objective neurologic findings were present; (4) the affliction came on following craniocerebral trauma; (5) operation disclosed atrophy of the fibers of the posterior root of the nerve, plus adhesions to both the dura propria and the nerve fibers themselves. Fortunately for this patient, posterior root section afforded complete relief; this had not been possible by alcohol injection.

Case II. *Atypical major trigeminal neuralgia in a middle-aged woman for seven years. Impaired sensation of the face, tinnitus, and loss of hearing on the affected side. Exploration revealed pressure grooving of the affected nerves from anomalous veins; electrocoagulation and resection of the veins gave complete relief.*

A 57 year old woman was admitted to the hospital (June 3, 1938) because of severe pain on the left side of her face. Seven years previously she had suffered the first attack of pain, located in the lower left side of the face and base of the tongue. Since that time she had had more or less continuous dull aching pain which had radiated through the area innervated by the left maxillary and mandibular divisions of the trigeminal nerve. Tingling had occurred in the upper lip on several occasions and had continued for two or three weeks at a time. During the attacks there were, in addition to continuous pain, frequent paroxysms of severe electric shock-like pain which occurred spontaneously or from stimulation of trigger zones on the lips. These paroxysms had recurred many times daily for periods of weeks or months. At the onset of facial pain, the patient was also aware of buzzing and deafness in the left ear; the deafness had steadily increased.

The woman had suffered from hyperthyroidism fifteen years before (1923) and had been relieved by thyroidectomy. Four years before (1934) she had begun to suffer from attacks of biliary colic; cholecystectomy the same year relieved this condition. She had also

suffered for twenty years from extensive varicose veins of the lower extremities.

Her state of nutrition was good; she was slightly obese. The blood pressure was 134 systolic and 88 diastolic; the pulse averaged 76 beats per minute; the temperature was 98° F. Extensive varicose veins in both lower extremities extended to the hips. The neurologic examination revealed a moderate hemihypesthesia of the face corresponding to the distribution of the maxillary and mandibular branches of the left trigeminal nerve, and a marked impairment of hearing on the left side. Barany's test on the left side gave a sluggish response from both the horizontal and vertical canals, and there was no constitutional reaction or past pointing. On the right side the findings were normal. The neurologic examination was otherwise negative. Roentgenograms of the skull disclosed no abnormality. The Wassermann reaction of both blood and spinal fluid was negative. Analysis of blood and urine revealed nothing abnormal.

On June 8, 1938, the left cerebellopontine angle was explored through a unilateral suboccipital approach. Both the trigeminal and acoustic nerves were almost completely obliterated from view by a large varix of the petrosal sinus. After these veins had been electrocoagulated they were resected; then both the acoustic and trigeminal nerves could be seen and showed definite pressure grooving. (Fig. 2.) Since an adequate cause for the symptoms was found and removed, the root of the trigeminal nerve was not sectioned.

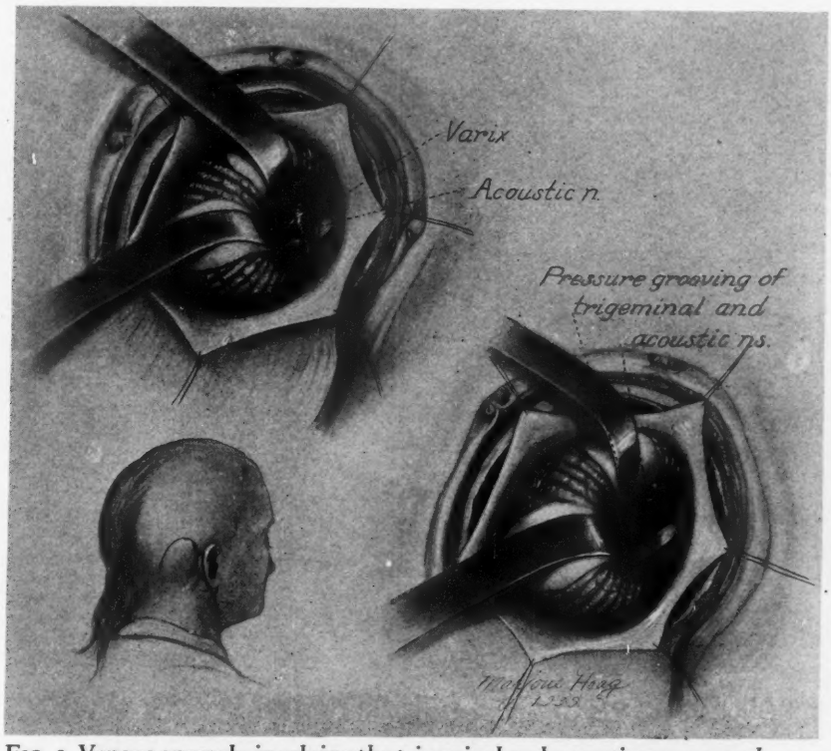


Fig. 2. Venous anomaly involving the trigeminal and acoustic nerves, and pressure grooving of the nerves observed after removal of the varix.

The day after operation, stroking of the face where trigger zones had previously existed failed to bring on an attack of pain, and the continuous pain had also disappeared. Buzzing in the ear had subsided, but impaired sensation of the face and loss of hearing were about the same as preoperatively. The patient was discharged from the hospital on the fourteenth postoperative day, completely free from pain. When last seen (December 9, 1938) six months after operation, there had been no return of symptoms. The impairment of sensation in the face had largely disappeared and hearing had markedly improved.

*Comment.* The illness of this patient whose illness was in some respects fairly typical of the classic type of major tri-

geminal neuralgia. The symptoms differed, however, in that dull aching facial pain was constantly present between the paroxysmal attacks, and objective neurologic findings were present. Because the physical findings led to a diagnosis of an organic lesion involving both the acoustic and trigeminal nerves (thought probably to be neoplastic) the suboccipital approach to the cerebellopontine angle was chosen, and this disclosed the unusual varix.

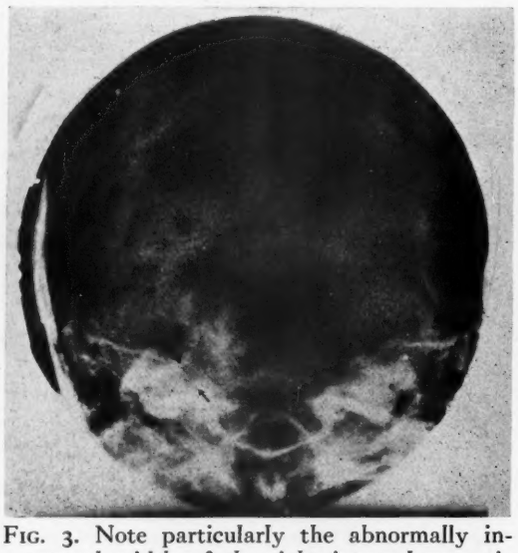

FIG. 3. Note particularly the abnormally increased width of the right internal acoustic meatus and the adjacent area of calcification.

The writer does not wish to postulate an increased tendency of varices to develop within the cranial cavity because of their presence to an extensive degree in the lower extremities. However, when an intracranial condition does develop, as in the case of this patient, extensive varicosities elsewhere in the body may possibly serve to indicate the nature of the lesion. Lastly, it was gratifying that both auditory and trigeminal nerve symptoms disappeared following destruction of the varix; thus, the necessity of a subsequent operation with consequent sacrifice of cranial nerve function was obviated.

CASE III. *Attacks of major trigeminal neuralgia and constant right facial pain for seven years in an elderly woman. Deafness in the right ear for nineteen years. Hypesthesia of the right side of face. Roentgenographic evidence of localized calcification at the right internal acoustic meatus. Fractional section of the posterior root of the right trigeminal nerve gave complete relief from symptoms.*

A 67 year old woman was admitted to the hospital August 8, 1934 because of attacks of severe pain in the right side of the face. She stated that the first attack had occurred seven years previously early one morning while she was brushing her teeth, and that for several weeks thereafter she had suffered from paroxysmal attacks of pain which radiated to the right side of the nose, teeth, and tongue. Since that time she had suffered from constant dull aching pain in the right side of the face, with exacerbations and remissions of the paroxysmal pain lasting several weeks at a time.

Further interrogation revealed that nineteen years previously (1919) she had suffered from continuous ringing in the right ear which had subsided after six months; then deafness was first noticed. By the end of three or four years she had become totally deaf in the right ear.

The patient was well nourished, with a blood pressure of 150/86, pulse of 70, and temperature of 98.4°F. The neurologic examination gave the following findings: (1) moderate hypesthesia through the distribution of all the branches of the right trigeminal nerve; (2) absence of the right corneal reflex; (3) trigger zones at the right nasolabial fold which when stimulated precipitated seizures typical of major trigeminal neuralgia, (4) total loss of hearing in the right ear. Barany's test on the right side produced no nystagmus from stimulation of either the vertical or horizontal canals, and there was no constitutional reaction or past pointing. On the left side there was no response from the vertical canals and only a slight nystagmus from the horizontal canal; there was no constitutional reaction and only slight past pointing.

Roentgenograms of the skull disclosed a circumscribed area of increased density approximately 2 cm. in diameter at the right internal acoustic meatus. (Fig. 3.) Spinal puncture gave a pressure of 70 mm. of water; the fluid was clear; there was one cell, and no increase in total protein. The Wassermann reaction of both blood and spinal fluid was negative. The blood count was within normal limits, and urinalysis gave normal findings. A calcified acoustic neuroma was presumably present, but had apparently been quiescent for a number of years. Several previous alcohol injections had been unsatisfactory. Permission for posterior root

section of the trigeminal nerve was refused though the patient begged for relief from pain.

Two years later (February 20, 1936) she was readmitted to the hospital for further observation and treatment. Examination at that time gave essentially the same findings as previously, with the exception that there was almost complete anesthesia in the area of distribution of the third division of the right trigeminal nerve (probably the result of alcohol injection). Barany's test was repeated and gave the same results as before. Roentgenograms showed the area of increased density (calcification) at the right internal acoustic meatus unchanged. Routine findings otherwise were essentially normal.

Six days later (February 26, 1936) consent for operation was obtained and the posterior root of the right trigeminal nerve was sectioned by way of the temporal approach. The dural incision also was extended farther posteriorly through the tentorium and the upper border of an encapsulated tumor (presumably of the acoustic nerve) came into view; it apparently was pressing on the root of the trigeminal nerve. (Fig. 4.) Later microscopic study of a biopsy revealed the characteristics of a perineural fibroblastoma.

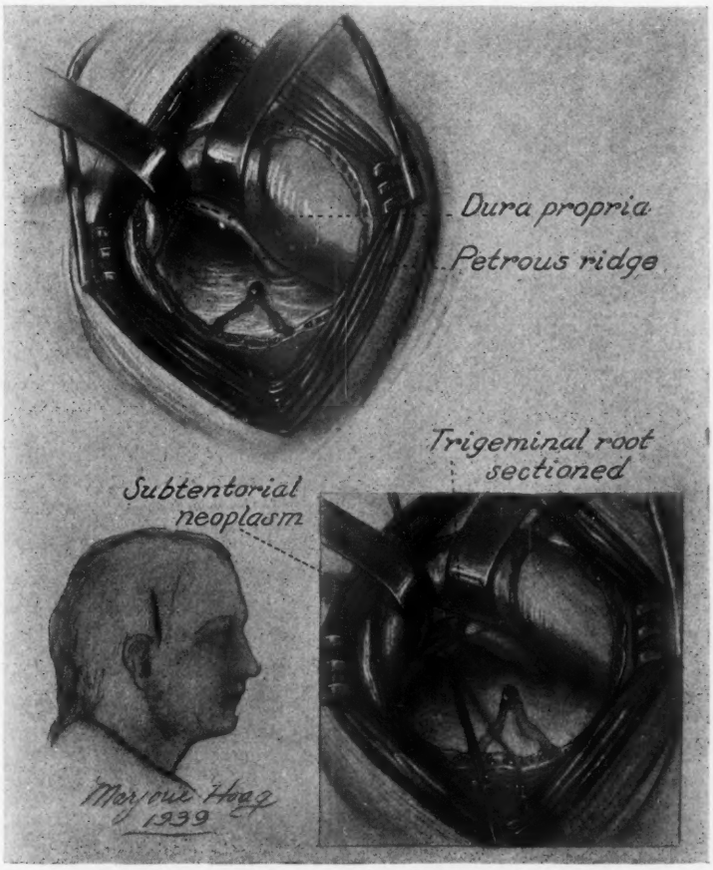


FIG. 4. Neoplasm of the posterior fossa (probably an acoustic neuroma) pressing on the posterior root of the trigeminal nerve.

Convalescence was uneventful and the patient was discharged from the hospital on the sixteenth postoperative day free from pain. When last seen two years after operation (October 9, 1938) there had been no return of pain; roentgenograms showed no increase in the area of density (calcification) near the right internal acoustic meatus, and there were no clinical signs of increasing intracranial pressure.

*Comment.* This patient's complaint on admission was primarily that of a typical case of major trigeminal neuralgia. However, continuous burning pain between the severe paroxysmal attacks and objective neurologic findings placed it in the atypical group. At operation a gross pathologic cause for the disturbance was found. The earlier symptoms of involvement of the acoustic nerve had never been a source

of complaint. In the initial history she actually denied tinnitus and deafness, but after the examination showed involvement of the trigeminal and acoustic nerves on the right side and also positive roentgenographic evidence of a lesion in the right cerebellopontine angle, she finally admitted having suffered from tinnitus and deafness twenty years before. In this atypical case it will again be noted that the pain was confined to the anatomic distribution of the trigeminal nerve.

Now the question might arise as to why an attack was not made upon the tumor rather than selecting the temporal approach for section of the root of the trigeminal nerve. Several factors determined the selectivity of the treatment employed: (1) the advanced age of the patient; (2) the absence of signs indicative of any increase in size of the tumor for several years; (3) the increased danger involved from complete removal of the tumor; (4) the complete facial paralysis that would have resulted from such a procedure; (5) the presence of pain was the patient's only reason for presenting herself for treatment.

Case IV. *A young woman who had suffered continuous pain and progressive loss of sensation in the distribution of the right trigeminal nerve for one year. Arrested pulmonary tuberculosis. Operation disclosed a chronic inflammatory process involving the dura propria, ganglion, and posterior root of the trigeminal nerve; section of the root gave relief from pain.*

The patient, a woman of 29 years, was first admitted to the hospital April 12, 1928 because of abdominal pain. A diagnosis of acute appendicitis was made, and the appendix was promptly removed. The pathologic examination, however, failed to show any inflammatory changes. The postoperative convalescence was uneventful.

The patient was again admitted to the hospital six years later (May 20, 1934), this time because of pain in the left shoulder. Examination revealed a moderately advanced pulmonary tuberculosis in the upper lobe of the left lung. She was then sent to Olive View Sanitarium where she was treated, and discharged six months later as an arrested case.

She was not heard from again until three more years had elapsed, at which time she was complaining of left facial pain. At the onset one year previously the pain had been confined to the left eye, but on readmission to the hospital (July 2, 1937) it had involved both the first and second divisions of the nerve; the pain also had increased in severity. There was no history indicating that trigger zones had been present, and there had been no paroxysmal attacks of pain, but there was increasing numbness of the face. The patient had lost considerable weight.

Impaired sensation in the first and second divisions of the left trigeminal nerve was noted. Roentgenograms of the skull showed no abnormality. The spinal fluid examination was not remarkable.

Four months later, October 8, 1937, the pain had become more severe and had involved all three divisions of the left trigeminal nerve. Because of severe pain on eating, an adequate diet could not be maintained. This resulted in a loss of 15 additional pounds. The area of impaired sensation had spread to all three divisions of the nerve. Spinal fluid examination again gave normal findings. Because of the progressive nature of the disease a diagnosis was made of an organic lesion (presumably neoplastic) involving the left trigeminal nerve.

Four days later the right Gasserian ganglion was explored. The dura in the region of the ganglion and posterior root was densely adherent to the base of the skull, and actually appeared to be covered with granulation tissue. Careful inspection failed to disclose a tumor. The dura propria was then opened, but no cerebrospinal fluid escaped. The root of the trigeminal nerve was yellowish in appearance, and approximately one-third the normal size; it was adherent to the dura, and the fibers themselves were adherent to one another. The appearance was that of a single cord. Exploration farther posteriorly was not done for fear of opening into the subarachnoid space, which was apparently walled off by adhesions. The entire posterior root of the nerve was sectioned. (Fig. 5.)

The postoperative convalescence was uneventful and complete relief from pain was obtained. Fourteen days after operation the patient was discharged from the hospital and had begun to gain weight. When last seen

seven months after operation, she had regained her lost weight; anesthesia on the right side of the face was complete, and there had been no return of pain.

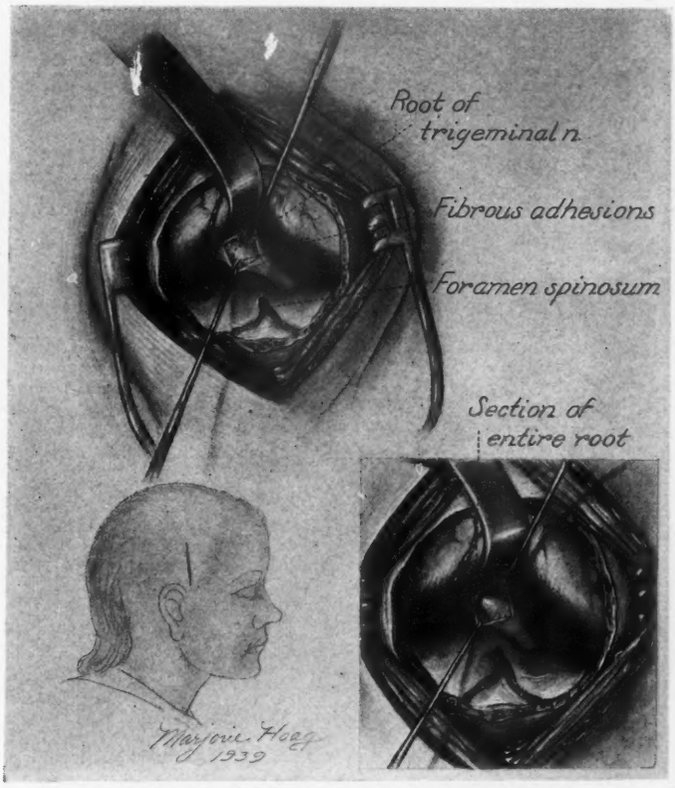


Fig. 5. Note the diminished size of the posterior root of the trigeminal nerve and adhesions to the dura propria. Also the confluence of the nerve fibers forming what appears to be a single cord.

*Comment.* Unfortunately in this patient the true nature of the inflammatory process involving the dura propria, Gasserian ganglion, and posterior root, was not disclosed; cultures and smears failed to reveal organisms. Of clinical interest, however, was the resemblance of the symptoms to those of a Gasserian ganglion tumor. At no time during the period of observations was there any indication that a low grade infection existed. On the other hand, there was nothing regarding this patient's illness that suggested major trigeminal neuralgia of the classic type, i.e., the pain was continuous and not paroxysmal, there were no trigger zones, and objective findings were present. Lastly, section of the posterior root gave relief.

Case v. *Attacks, in a middle-aged woman, of major trigeminal neuralgia and tinnitus on the left side for twenty years. Barany's test gave findings suggestive of an organic lesion involving the left acoustic nerve. Operation revealed an acoustic neuroma exerting pressure on the root of the left trigeminal nerve. Death occurred the day after operation from coronary thrombosis.*

A 54 year old woman was admitted to the hospital September 1, 1937 because of recurring attacks of pain in the left side of the face. She had been suffering from pain in the left side of the face for the previous twenty years. The onset of the first attack, like all the subsequent attacks, was sudden; it came on while she was drinking a glass of cold water, and passed away as quickly as it had appeared; it radiated to the angle of the jaw and the lower teeth. For several months thereafter drinking cold water or partaking of cold food precipitated attacks. The following two or three years she was free from pain, and then the attacks again recurred, this time for a longer period.

When admitted to the hospital, she had been suffering almost continuously for three years; furthermore, the pain had spread to the maxillary branch of the nerve. No trigger zones had been present.

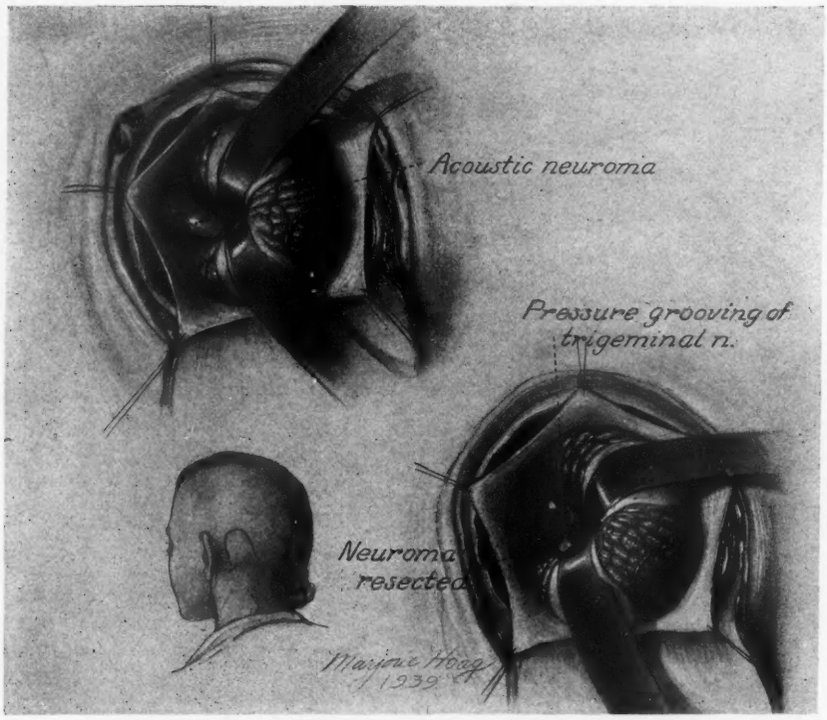


FIG. 6. Small neoplasm of the acoustic nerve and pressure grooving of the trigeminal nerve that could be seen after removal of the tumor.

The patient had suffered the usual childhood diseases, and had gone through three normal pregnancies. The children were all living and well. A hysterectomy was done in 1929 for uterine fibroids.

Further interrogation revealed occasional attacks of ringing in the left ear, which had lasted two or three weeks at a time. She further stated that ringing in the ear had begun prior to the onset of facial pain, but that it had never been a source of discomfort. Alcohol injection of the maxillary and mandibular branches of the affected nerve had given relief for short periods of time, but the last two injections had been unsuccessful.

Examination showed the patient to be well developed, and in a good state of nutrition. The blood pressure was 134/78, the pulse 70, the temperature 98.2°F. Neurologic examination was negative except for the trigeminal and acoustic nerves on the left side.

There were patchy areas of hypesthesia in the maxillary and mandibular branches of the left trigeminal nerve (probably due to previous alcohol injections). Hearing tests for the ordinary range of sounds were normal. Barany's test on the left side gave no constitutional response or past pointing; only a few coarse, perverted, nystagmoid movements appeared after stimulation for four minutes of both vertical and horizontal canals; on the right side the findings were normal. The Wassermann reaction of both blood and spinal fluid was negative. The spinal fluid pressure was 120 mm. of water; there was one cell per c. mm. and no increase in globulin. Urine and blood examinations were not remarkable. Examination otherwise gave normal findings. Because of the history of tinnitus and the findings obtained from Barany's test, a probable diagnosis of acoustic neuroma was made.

The left cerebellopontine angle was explored September 3, 1937 by way of the suboccipital route. A small encapsulated tumor of the acoustic nerve was found, and removed without technical difficulty. After removal of the tumor the root of the trigeminal nerve was observed to have been flattened by the tumor on its inferior and lateral surfaces. (Fig. 6.) Since a reasonable cause for the neuralgia was found and removed, the root of the trigeminal nerve was not sectioned.

The day after operation the patient had recovered from the effects of anesthesia and was in good condition. The blood pressure was 110 systolic and 82 diastolic; the pulse averaged 100 beats per minute; the temperature was 99.8°F. In the evening she partook of a light supper and felt reasonably well; there were no attacks of neuralgia. Two hours later the nurse discovered the patient was dead.

Post-mortem examination revealed moderate general arteriosclerosis; the coronary arteries showed atheromatous changes and recent extensive thrombosis. Examination of the brain except for the ordinary amount of operative trauma was not remarkable.

*Comment.* This patient, like the others, complained chiefly of pain referable to the anatomic distribution of the affected trigeminal nerve; the nature of the pain, however, differed from the others in that it resembled the pain of classic trigeminal neuralgia. The associated atypical features of tinnitus and Barany test findings already described need no further elucidation.

While the pain varied with each case reported and was quite different in some from the pain in the classic type of Fothergill's disease, it will be noted that in all cases it was confined to the anatomic distribution of the affected trigeminal nerve. Furthermore, there were physical signs of organic disease of the trigeminal nerve itself or other regional cranial nerves. If these points are kept in mind the proper selection of cases for surgical treatment should be materially facilitated. Lastly, surgery gave uniform relief and the findings at operation were consistent with the subjective complaints and the objective neurologic signs.

## SUMMARY

1. Pathologic causes of major trigeminal neuralgia frequently cannot be demonstrated in both the typical and the less clearly defined (atypical) cases.

2. In the classic type of the disorder (Fothergill's disease), relief is most satisfactorily obtained by surgery. In the atypical syndromes surgery has generally been considered to be contraindicated; however, there are notable exceptions.

3. From the literature it appears that trigeminal neuralgia may have a variety of causes: (1) infections, (2) ill-defined sclerotic changes, (3) neoplasms, (4) vascular anamolies, etc.

4. Any attempt to classify the disease, aside from the classic and atypical forms, is almost impossible. However, two general groups can be recognized: (1) those which can be expected to respond to surgery; (2) those which cannot be expected to respond.

5. In those atypical cases in which relief by surgery may be anticipated, the pain generally follows the distribution of one or more branches of the trigeminal nerve, and neurologic findings may or may not be present.

6. In the cases reported a gross cause for the neuralgia was found which was consistent with the subjective complaints and the objective neurologic findings; relief was obtained by surgery, i.e., either by section of the posterior root of the trigeminal nerve or by removal of the cause.

## REFERENCES

1. Fothergill, John. Works. London, 1784. Chas. Dilly. Of a painful affection of the face. *Med. Observ. & Inquiries*, 5: 129, 1773.
2. Lewy, F. H. First authentic case of major trigeminal neuralgia and some comments on the history of this disease. *Ann. M. Hist.*, 10: 247, 1938.
3. Spiller, W. G. and Fraizer, C. H. The division of the sensory root of the trigeminus for relief of tic douloureux; an experimental, pathological and clinical study, with a preliminary report on one surgically successful case. *Phiiadelpbia M. J.*, 8: 1039, 1901.
4. Sjöqvist, Olof. Studies on Pain Conduction in the Trigeminal Nerve. New York, 1938. G. E. Stechert.
5. Adrian, E. D. and Zotterman, Y. The impulses produced by sensory nerve-endings. II. The response of a single end-organ. *J. Physiol.*, 61: 151, 167, 1926.
6. Gasser, H. A. and Erlanger, J. The role played by the size of the constituent fibers of a nerve trunk in determining the form of its action potential wave. *Am. J. Physiol.*, 80: 522, 1927.
7. Parker, H. L. Trigeminal neuralgic pain associated with multiple sclerosis. *Brain*, 51: 46, 1928.
8. Dandy, W. E. Cause of trifacial neuralgia. *Am. J. Surg.*, 24: 447, 1934.

9. Alajouanine, R. and Thurel, R. Algies faciales posttraumatiques. Guérison par anesthésie du ganglion sphéno-palatin. *Rev. neurol.*, 1: 404, 1937.
10. Colucci, G. Su di un raro caso di nevralgia del trigemino di origine traumatica. *Riforma med.*, 52: 49, 1936.
11. Aubertine, D. J. Etiologic factors in tic douloureux. *J. Am. Dent. A.*, 23: 2271, 1936.
12. Hoff, H. Zwei Fälle mit besonderer Tuberkulinreaktion. *Wien. med. Wchnschr.*, 84: 811, 1934.
13. Harris, W. Paroxysmal neuralgic tic as a sequel of trigeminal neuritis. *Brit. M. J.*, 1: 1112, 1935.
14. Hevia, A. R. Estudio fisiológico en la neuralgia esencial del tregemino. *Rev. méd. de Chile*, 65: 794, 1937.
15. Lewy, F. H., and Grant, F. C. Physiopathologic and pathoanatomic aspects of major trigeminal neuralgia. *Arch. Neurol. & Psychiat.*, 40: 1126, 1938.

There can be no dogmatic advice with regard to patients suffering from trigeminal neuralgia as to which form of treatment is the best; every patient must be considered independently.

# EPIPHYSEAL CHANGES RESULTING FROM ACUTE INFECTIONS OF BONE

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THIS study of epiphyseal changes associated with acute hematogenous osteomyelitis was initiated partly because of the large number of records accessible at The Mayo Clinic concerning patients with chronic and acute osteomyelitis. It was felt that the series was large enough to permit the drawing of some conclusions and that the group would represent a cross section of patients who had received various types of treatment from surgeons over a large geographical area. It was hoped to ascertain the frequency of deformities and their variety.

Another reason for the present study was the too frequent use of the word "epiphysitis." In reality, few lesions ever can be demonstrated in the epiphysis and, over a period of years, one epiphysis does not demonstrably differ from its fellow. Experienced surgeons are alert to discount a report that epiphysitis, with subsequent disturbance of growth, resulted from the common osteomyelitis. The young surgeon, however, who has not had long experience in treating this condition and observing the results, may be misled by the general impression that epiphysitis is common.

A report of the survey of literature which preceded the study on which this paper is based was included in an unpublished thesis.[1] For consideration of space it is omitted here.

## MATERIAL

To secure material for study, I reviewed the records of 2,064 patients with acute and chronic osteomyelitis encountered at The Mayo Clinic from 1930 to 1937 inclusive. In any case in which the history or the report of examination suggested the presence of epiphyseal changes, the record was checked against the roentgenograms. Further study was given only to those cases in which there was definite roentgenologic evidence of epiphyseal change. Cases in which there were changes in joints and closure of the epiphyseal line as a result of infections of low grade, without the usual acute symptoms of hematogenous osteomyelitis and cases of acute traumatic osteomyelitis, with associated epiphyseal changes, were excluded. Cases in which there was lengthening of extremities, without evidence of demonstrable changes in the epiphysis or epiphyseal line, were not included. In many cases in which lengthening of an extremity takes place, the pathologic lesions are too far distant from the epiphyseal line to be considered results of epiphyseal stimulation or irritation; the lengthening possibly results from increased circulation in the diaphysis adjacent to the epiphyseal line.

Of this group of 2,064 cases, sixty-six showed both clinical and roentgenographic evidences of epiphyseal changes following an osteomyelitic condition.

## RESULTS

*Upper Femoral Epiphysis.* In twenty-seven cases a hip joint was involved. In this, the largest group, results were consistently poor. There was no instance in which the epiphyseal line had been spared. In one case, however, there was evidence of a remaining epiphyseal line but it was narrow and abnormally developed in comparison with that of the opposite hip; the epiphysis was flattened and thinner

than normal and there was some irregularity of contour, particularly on the weight-bearing aspect.

FIG. 1. Characteristic changes in hips after early incision and drainage. Absorption of the epiphysis and a portion of the neck of the femur.

FIG. 2. Preservation of the epiphysis and bony ankylosis.

In one case, in which treatment had been conservative and roentgenologic examination was made frequently, there was gradual absorption of the juxta-epiphyseal tissue. This was followed by absorption of the epiphyseal cartilage and, at the end of the third month, the epiphysis displayed the characteristics of a sequestrum. Later a sequestrum was removed.

In two cases in which treatment had been conservative pathologic dislocations were present and the epiphyseal lines were closed. The epiphysis or femoral head in one case was intact, although it was smaller than the femoral head on the other side; in the second case the portion of the head adjacent to the ilium had undergone absorptive changes and irregularity of the epiphysis resulted.

In one case, that of an infant nine months of age, there was evidence of complete absorption of the neck of the femur but the epiphysis appeared normal on roentgenologic examination.

Absorption of the epiphysis and a varying degree of absorption of the neck of the femur were evident in four cases. Treatment in all four was by early incision and drainage; there was no evidence of ankylosis. (Fig. 1.)

In two cases, the epiphysis was absorbed without evidence of change in the juxta-epiphyseal region. In neither case was there ankylosis. Treatment had consisted of early incision and drainage.

In one case, the epiphysis and the juxta-epiphyseal region were preserved without ankylosis, but destructive and hypertrophic changes had taken place in the head. There was associated change in the acetabulum. Treatment had consisted of early incision and drainage.

In twelve cases, ankylosis was present but part or all of the epiphysis and juxta-epiphyseal region were preserved. In ten cases treatment had been conservative and in two, early incision and drainage had been carried out. (Fig. 2.)

In two cases in which the epiphysis was preserved, ankylosis followed incision and drainage for acute osteomyelitis of the greater trochanter and ilium, respectively.

Another case presented the characteristics of acute inflammation of the upper femoral epiphysis, resulting from osteomyelitis of the neck of the femur; drainage had been instituted late in the course of the disease. There was marked destruction of the epiphysis and the juxta-epiphyseal region without ankylosis.

In this group of twenty-seven cases, the age of onset ranged from 7 to 19 years with the exception of the case of the infant of nine months. Six patients were aged 9 years, twelve were aged 8 years and four, 14 years. The remaining cases were distributed over the other years as single cases or in groups of two.

*Lower Femoral Epiphysis.* This group, composed of fifteen cases, constituted the next to the largest in the series.

FIG. 3. A, involvement of the lower femoral epiphysis; eighteen days following incision and drainage; spotted absorption of the medial epiphyseal line; B, four months following onset; beginning recalcification of the affected portion, with a large region of absorption in medial portion of the epiphysis; C, ten months following onset; closure of the medial femoral epiphyseal line, especially along the periphery; early genu valgum.

In two of the cases the condition was of recent origin and the patients were seen at the clinic early in the course of the disease. In one of these cases, which was of five weeks' duration, there was evidence of absorption in the cartilaginous plate of a medial femoral epiphysis; the absorptive changes had followed acute arthritis of the knee. Drainage had not been instituted. There was no record of late follow-up examination. In the second case, that of a child of 8 years, the history was that the condition had begun acutely, with swelling in a knee, in October 1935. Seven days following this, aspiration of the knee was performed and the periosteum was incised on the medial aspect of the lower part of the femur. On the eleventh day following onset roentgenologic changes were noted. Twenty-nine days following onset and eighteen days following incision and drainage, roentgenologic examination gave evidence of spotted absorption, involving the medial epiphyseal line, and absorption of the cartilaginous disc. Four months from the time of onset, examination revealed recalcification of the affected portion of the metaphysis, with absorption of the medial epiphyseal line; the absorption extended into the epiphysis. Eight months following onset, more evidence of calcification was observed; there was a residual lesion resembling a cavity in the metaphysis and extending into the epiphysis. Also there was beginning shortening of the medial aspect of the femur. On clinical examination, ten months after onset, definite shortening and outward bowing were perceptible. Two years following onset, the bone appeared to have healed with complete closure of the medial aspect of the epiphyseal line. (Fig. 3.)

The remaining thirteen patients of this group were seen late in the course of the disease. In three cases there was primary septic arthritis of a knee; in two of these early incision and drainage had been carried out. In one case of the three a genu valgum deformity resulted; in the second case there was ankylosis, with incomplete fusion of the epiphyseal line anteriorly, and consequent overgrowth of the anterior portion of the epiphysis. In the third case, actual shortening of 3 inches (7.5 cm.) resulted from closure of the femoral epiphyseal line. The onset had been at the age of 11 years and the condition had

existed for fourteen years. There was no deformity from deviation and the functional result was fair.

FIG. 4. Anteroposterior view showing a defect in the lateral femoral epiphysis with an associated genu valgum deformity.

In nine of the remaining ten cases the primary focus of infection was felt to be metaphysitis. The epiphyseal changes in the tenth case may have resulted directly from metaphysitis or from septic arthritis of a knee joint, since the knee was secondarily infected following incision and drainage for metaphysitis. The deformity was of genu valgum type and probably resulted from injury to the epiphysis at the time of the original surgical intervention. (Fig. 4.)

Of the nine cases, three presented genu valgum deformity from acute metaphysitis, with associated change in a medial femoral epiphysis. In two of this group of nine cases, early incision and drainage had been done for acute metaphysitis with subsequent epiphyseal changes; varus deformity resulted. Four of the nine cases presented shortening as a result of femoral epiphyseal changes, without deformity from deviation. In two of these four cases drainage was not instituted and shortening was of 1 inch (2.5 cm.) and of ½ inch (1.25 cm.), respectively. The onset in the first of these two cases had been at the age of 14 years and the condition had endured twenty-one years; in the other of the two cases, onset was at the age of 12 years and duration of the condition had been four and one-half months. In the two remaining cases of the four, incision and drainage had been effected early; in one, shortening was 3½ inches (9 cm.) and in the other, 10 inches (25 cm.). In the first of the two cases just mentioned, onset had occurred at the age of 17 years and in the second at the age of 5 years; in both cases the duration of the condition had been fifteen years.

*Distal Tibial Epiphysis.* Involvement of the distal tibial epiphysis comprised the third group in point of numbers of cases; there were eight. In two cases onset was in infancy, in two, before the fifth year, and in the remaining four, at ages 9, 12, 13 and 14 years, respectively. In this group there were several interesting epiphyseal changes.

Two of the patients presented ankylosis of the ankle. In one of these two cases there was lengthening of a tibia; probably the ankle joint was infected secondary to surgical drainage of the focus. In the other case in which ankylosis occurred, there was closure of the epiphyseal line and ⅜ inch (1 cm.) of shortening. Drainage had been instituted early and, at the time of examination, the disease had existed for three years.

In two cases varus deformity was present. In one, duration of the disease had been fifteen years from onset at the age of 4 years. In this case drainage had been effected and the bone had been scraped early in the course of the disease; shortening of the tibia by 1¼ inches (3 cm.) resulted. (Fig. 5.) In the second case in which varus deformity was present, the condition had existed for one year after its onset at the age of 14 years; absorption of the cartilage of the epiphyseal line and a residual, thin, smooth line remained. Incision and drainage had been effected early.

Two patients presented a calcaneal deformity; one, seen at the age of 13 years, after the condition had existed thirteen years, had undergone early incision and drainage and presented an unusual deformity. There was a defect in the epiphysis and epiphyseal line of the tibia and the head and neck of the corresponding talus were separated from its body and were occupying the defective space in the epiphysis and epiphyseal line. There was 5/8 inch (1.5 cm.) shortening of the extremity with lateral deviation. The second patient who presented calcaneal deformity was seen at the age of 9 years, after the condition had existed for six years. Drainage had been instituted at five and fifteen days, respectively, following the onset of acute symptoms. Examination disclosed that destruction of the medial epiphyseal line had taken place and that the distal end of the fibula was at the lower level of the tarsal bones. There was 1¾ inches (4.5 cm.) of shortening of the extremity, without lateral deviation.

Fig. 5. Lateral and anteroposterior views showing shortening of the tibia with varus deformity as a result of epiphyseal involvement early in childhood.

The two remaining patients presented unusual conditions. The disease of both had begun in infancy. One, aged 7 years, had undergone early incision and drainage and roentgenologic examination disclosed two lines in the lower part of the tibia, each with the characteristics of an epiphyseal line. There was a lesion in the posterior aspect of the tibia which extended through what appeared to be the proximal epiphyseal line and to the normally placed epiphyseal line; the lesion involved the adjacent portion of the epiphysis. The proximal one of the lines, which had the characteristics of an epiphyseal line, represented a growth line, described some time ago, and again more recently by Siegling. He presented roentgenograms of infants after they had ingested phosphorized cod liver oil; growth lines were clearly demonstrated. The remaining of the two patients whose condition had begun in infancy was seen at the age of 5 years. At that time there was extensive osteomyelitic involvement of the shaft of the tibia, absorption of the lower tibial epiphyseal line and the epiphysis presented the roentgenologic characteristics of a sequestrum.

*The Proximal Tibial Epiphysis.* Involvement of a proximal tibial epiphysis was noted in six cases.

Three of this group of patients presented a genu valgum deformity; two of

the three had been subjected to early incision and drainage and the third to the making of multiple drill holes for drainage.

FIG. 6. Anteroposterior view showing sclerotic change in epiphyseal line.

One of the three patients, seen at the age of 10 years, after the condition had existed for two years, presented, on roentgenologic examination, a defect of the epiphysis which was suggestive of a surgical defect. There were absorptive and destructive changes in the anterior epiphyseal line and sclerosing changes in the posterior aspect of the epiphyseal line. The second of these three patients, seen at the age of sixty-four years, gave an indefinite history of an acute infection of bone at an early age. This was drained early and a genu valgum deformity resulted. The third patient of the three, treated by early drilling, was seen at the age of 5 years, after the condition had endured for two years. In this case perimetric hypertrophy of the tibia was present; the epiphyseal line remained open but there were some sclerotic changes. There was actual lengthening of 1 inch (2.5 cm.).

In two cases there was no deviation deformity. One of these two patients, seen at the age of 10 years, after the condition had existed for three years, presented involvement of the metaphyseal plate and the corresponding portion of the epiphysis

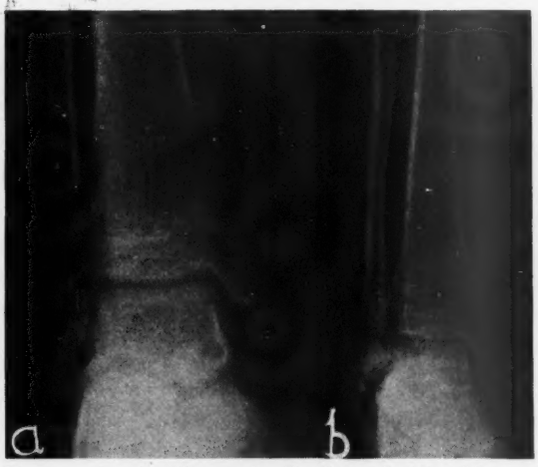


FIG. 7. A, early change in diaphyseal side of lower fibular epiphysis; five weeks after drainage of soft tissue abscess; absorptive changes in fibular epiphysis; a region of the tibial epiphysis is suggestive of absorption. B, fourteen months after onset; fibular and tibial epiphyseal lines of same development; residual scarring epiphyseal line of tibia; fibular epiphysis regenerated, save distal tip.

was growing into the defect. Surgical operation had not been performed. The second of the two patients was aged 13 years and the condition had existed for one year. There was lengthening of ½ inch (1.25 cm.) and sclerosing changes involved the entire epiphyseal line (Fig. 6).

In the final case, that of a patient aged 22 years, whose condition had existed for five years, drilling for drainage had been done early after onset. Ankylosis of the knee, a genu valgum deformity and 6 cm. of shortening were present.

*Proximal Humeral Epiphysis.* This group consisted of four cases, one of which is described in the section on multiple epiphyseal involvement. The second case, that of a child of 11 years, whose condition had existed for six months, had undergone early drainage; absorption of the upper humeral epiphysis had taken place and destructive changes of the epiphysis were present. In the third case, that of a patient 18 years of age, whose disease had been present for seven years, closure

of the proximal humeral epiphysis and upward growth of the greater tuberosity were evident. Loss of the angle of the surgical neck of the humerus had resulted. The fourth case, that of a patient of 9 years, whose disease was of one year's duration, had been subjected to early drainage; there were involvement of the epiphyseal line and destructive changes in the epiphysis.

*Multiple Epiphyseal Lesions.* Two patients presented multiple epiphyseal lesions. The disease of one of these, who was seen at the age of 30 years, had begun at the age of seven years with metaphysitis of the tibia. Drainage had been instituted late. Untoward developments were as follows: secondary infection of a knee and apparently subsequent involvement of the corresponding medial femoral epiphysis; resulting genu valgum deformity and increase in length of 1½ inch (4 cm.).

The second patient, aged 13 years, was seen at the time of onset and presented osteomyelitis of the proximal end of one humerus. Drainage was instituted. Results were closure of the epiphyseal line, continued growth of the epiphysis of the greater tuberosity and increase in the angulation of the surgical neck of the humerus. In addition, there was acute epiphysitis involving one hip joint; this resulted in closure of the epiphyseal line, destructive changes in the epiphysis and ankylosis. Surgical drainage was not instituted.

*Lower Fibular Epiphysis.* In this group of two cases the best end result of epiphyseal involvement of a long bone was encountered. One patient, aged 13 years, whose symptoms were of short duration, had been subjected to drainage of a superficial abscess on the twentieth day after onset of symptoms. The second patient, aged 12 years, was subjected to drainage of a soft tissue abscess on the eighth day after onset of symptoms. Both of these patients presented destructive changes in the lower epiphysis and diaphysis of one of the fibulas, with resulting complete reformation of the epiphysis. In the latter case, at the end of five weeks, roentgenologic examination (Fig. 7A) gave evidence of destructive changes in the diaphysis and epiphysis, with associated involvement of corresponding tibial epiphysis. Roentgenologic examination at the end of ten weeks disclosed involvement of the entire shaft of the fibula; apparently, also, there was a lesion in the corresponding lower tibial epiphysis and epiphyseal line. Roentgenologic examination at the ninth month gave evidence of regenerative changes in the fibular epiphysis and of healing of the lesion in the tibial epiphysis. At the end of fourteen months roentgenologic examination gave evidence of regeneration of the fibular epiphysis and there was no residual evidence of the lesion of the tibial epiphysis. (Fig. 7B.)

In addition to the case just reviewed in which healing of an epiphyseal lesion was evidenced both by clinical and by roentgenologic examination before the age of 13 years, in two cases which were studied but were not included in the series, there was involvement of one epiphyseal line of a femur with complete healing and no residual deformity or changes in length. One of the patients, aged 9 years, was found to have a lesion involving the lower epiphyseal line of one femur but, on examination seven years later, changes in the epiphyseal line were not disclosed. On the other hand, chronic osteomyelitis was present in the middle third of the shaft of the femur. In the second case, that of a patient, also aged 9 years, whose symptoms had endured for one year, roentgenologic examination gave evidence of involvement of the metaphysis and lower femoral epiphysis on one side, with absorption of the medial femoral epiphyseal line. The epiphyseal line appeared to be fused in its central portion. Five years later there was no deformity or change in length of the affected extremity as compared with the opposite extremity.

*Acetabular Epiphysis.* In this series, two patients, aged 11 and 12 years respectively, presented destructive changes of the acetabulum on one side. The illness of the former patient had existed two years and

that of the latter patient, four months. The acetabulum of the patient whose illness had endured two years had elongated. There

FIG. 8. Result of acute acetabular epiphysitis; destructive changes in acetabulum with closure of epiphysis.

had been drainage at the end of eighteen months in this case, but in neither of the two cases was there roentgenologic evidence of change in the corresponding proximal femoral epiphysis. In both cases the epiphysis of the affected acetabulum was closed. (Fig. 8.)

*Distal Radial Epiphysis.* In one case a lower radial epiphysis was involved. The patient was 15 years old and the duration of the condition was two and one-half years. Incision and drainage had been effected early in the course of the disease. Besides a lesion in the epiphysis, there was involvement of the diaphysis and epiphyseal line. However, the deformity and evidence of fusion of the epiphyseal line were lacking. The cartilage of the epiphyseal line was of increased density.

*Metacarpal and Phalangeal Epiphyses.* This group is composed of two cases. In the first case, in which there were multiple osteomyelitic lesions, the epiphysis of the middle phalanx of the third finger was involved and there was associated osteomyelitis of the proximal phalanx. The lesion in the shaft of the middle phalanx resembled a cyst. The adjacent portion of the epiphysis also presented the characteristics of absorption and apparently the lesion in the shaft was continuous with the lesion in the epiphysis. There was associated narrowing of the proximal interphalangeal joint.

In the second case, there were also multiple osteomyelitic lesions, involvement of the epiphysis of a first metacarpal and associated deviating deformity of the thumb of the same hand.

In neither case was a surgical procedure carried out. In the second case, there was associated involvement of the proximal epiphysis of the corresponding humerus; this has been described previously.

## COMMENT

The occurrence of epiphyseal changes associated with infections of long bones is rare, except in those cases in which surgical trauma to the epiphysis results from incision and drainage. The exception to this statement is the occurrence of epiphysitis in the proximal femoral epiphysis, the distal femoral epiphysis and possibly the distal humeral epiphysis. Usually the epiphysitis occurs in these situations because the epiphysis is anatomically included in the joint.

The occurrence of epiphysitis in structures other than those which enter into the formation of a joint and other than those which are not traumatized in the course of surgical operation, probably results from trauma to the epiphyseal disc or epiphyseal plate before the onset of acute symptoms; that is, probably the region of the metaphysis is traumatized and the associated epiphyseal cartilage is injured; metaphysitis develops and the traumatized cartilage undergoes degenerative changes and may be subjected to infection by direct extension. Another possible source of epiphyseal change is stimulation or irritation from a sequestrum lying adjacent to changes in the epiphyseal line.

Involvement of an epiphyseal line and adjacent epiphysis may undergo healing without residual deformity or change; on subsequent roentgenologic examination the epiphysis and epiphyseal line may appear normal and there may be no clinical evi-

dence of disturbance of growth. Also, lesions in the epiphysis may remain over a long period without destructive or deforming changes; in the presence of lesions of this type the conclusion must be that the cause of the condition is blood-borne infection, organisms being deposited in the epiphysis with resulting degeneration and absorption.

Involvement of the epiphyseal line may result in one of two conditions. The first of these may be a process of absorption, resulting in partial or complete closure, and it is in this type of involvement that shortening and deformity usually result. In the presence of the second type of involvement, roentgenologic examination gives evidence that the epiphyseal line is of increased density and is broader than normal. This results from deposition of calcium in the cartilaginous disc. Deviation deformity is not usually associated with this type of epiphyseal change; lengthening of the bone commonly occurs. However, it should be stated that lengthening of an involved bone may result from hyperemia, although clinical or roentgenologic evidence of epiphyseal involvement cannot be demonstrated during the course of infection and the period of healing.

From roentgenologic evidence in two cases of this series it was apparent that in each a fibular epiphysis was involved and regeneration subsequently took place. Such behavior of an epiphysis of any other bone was not seen and it is barely possible that the absorptive changes may have resulted from hyperemia in association with the inflammatory process. However, this was not considered probable since the epiphyseal cartilage appeared to be absorbed and the avascular plate should not be associated in an osteoporotic process. In conjunction with this observation it was also interesting to note that in two cases in which pathologic dislocation of a hip resulted from acute epiphysitis, the usual destructive changes of the epiphysis were not found. Furthermore, in one case of acute epiphysitis of a proximal femoral epiphysis in which treatment consisted of five weeks of traction and of application of a cast, without incision or drainage, there occurred very slight destructive change in the epiphysis and subsequently ankylosis occurred with the extremity in good functional position. On the basis of the observation of regeneration of fibular epiphysis in two cases, of diminished or absent destruction of the femoral epiphysis with dislocation of the hip, in two cases, and of preservation of the epiphysis in one case in which treatment was by traction, it is suggested that pressure may play some part in the amount of destruction and absorption of an epiphysis.

In regard to the proximal femoral lesions, it was concluded that if treatment was conservative, that is, if early incision and drainage were omitted, but immobilization and traction were employed and ankylosis resulted, the best clinical end results were attained.

In regard to the tibial epiphysis, it is strongly suggested that epiphyseal changes result from surgical insult at the time of surgical drainage. However, changes may occur in those cases in which the tibial epiphysis is included in the knee joint. This view is supported by Johnston (1936); he reported complete or incomplete interruption of growth in 55.5 per cent of his series of cases in which treatment consisted of making multiple drill holes in the metaphysis, the holes extending to the diaphyseal side of the epiphyseal line.

It was shown further that the epiphysis may make marked physiologic response in an attempt to minimize or prevent deformity. This response takes the form of increased growth or hypertrophy of a portion of an epiphysis.

In two cases there was an unusual response of the talus in an attempt to fill in and occupy a lower tibial defect which had resulted from complete absorption of part of the lower tibial epiphysis. In both cases there was a calcaneal deformity of the foot.

The epiphysis may act as a sequestrum, although this is rare. If it does act thus,

surgical removal of the sequestrum is required.

In the course of acute metaphysitis the surgeon should guard against mistaking associated synovitis of the joint for primary suppurative arthritis. Drainage of such a joint may cause suppurative arthritis to develop, primarily or secondarily, from the metaphyseal focus and epiphyseal changes may result.

REFERENCE

1. Macey, H. B. Epiphyseal Changes Resulting from Acute Infections of Bone. Rochester, Minnesota, 1939, 62 pp. (Cloth bound thesis in manuscript on deposit in Library of The Mayo Clinic, Rochester, Minnesota.)

Congenital affections of the knee are mainly connected with the patella, which is sometimes absent, and then other deformities are associated with its non-development.

# URETEROCELE

## TREATMENT BY TRANSURETHRAL RESECTION

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URETEROCELE is not an uncommon finding in urologic practice. The term applies to cystic dilatations of the intramural portion of the ureter protruding into the bladder, which are the end result of either acquired or congenital partial occlusion of the ureteral orifice. It is to be distinguished from the uncommon prolapse of the ureter through the ureteral orifice, for in the latter the intravesical covering of the mass is ureteral mucosa, while bladder mucosa covers the intravesical portion of the ureterocele. All degrees of dilatation are encountered, from the very small, almost insignificant, lesions to huge sacs filling much or all of the bladder.

The recent contributions of Gutierrez, Vermooten, Lazarus, Huth, Rhodes, Hunner, Foley, Riba, Gibson, O'Conor and Johnson, and Dawley, among others, adequately review the rather extensive literature upon this subject and include comprehensive discussions of the etiology, anatomy, pathology, symptomatology and treatment. It would seem superfluous to present a repetition of these many excellent articles. Only a brief review of the subject, pertaining particularly to etiology and treatment, will be attempted. The accompanying case report seems of interest because of the excellent postoperative end results obtained through the use of a new type of transurethral treatment. This procedure is described.

*Pathology.* A ureterocele is usually a thin-walled, intravesical, cystic dilatation of the intramural portion of the ureter, and as such is composed of bladder mucosa on the vesical side and of ureteral wall on the inner side. Varying amounts of muscle and fibrous tissue are present. If infection exists, or if excessive fibrous or muscle tissue is present, the cyst wall appears thickened and opaque instead of thin, translucent and easily punctured. The cysts may vary from very small, insignificant structures to very large cysts filling the bladder. Occasionally the large cysts produce urinary retention by obstructing the vesical neck, and if extruded through the urethra they may become strangulated. The degree of ureteral and renal pathology encountered may range from little to severe damage accompanied by advanced hydroureter and hydronephrosis due to the obstruction and back pressure. In late cases, the musculature of the bladder about the ureter, which forms the intramural portion, is frequently damaged by the dilation to the extent that the ureterovesical valve is no longer competent and ureteral reflux occurs after the ureterocele is removed.

*Symptomatology.* Symptoms of ureterocele pertain to the upper urinary tract due to obstruction of the terminal ureter, and to the bladder as a result of either urethral obstruction by large cysts or secondary cystitis. Ureteral obstruction usually causes periodic, dull, aching pains in the loin, although occasionally they may be colicky in nature. These symptoms are similar to and not distinguishable from those noted in other types of ureteral obstruction. Usually the patient endures the mild symptoms, but when severe symptoms incident to superimposed infection occur later, he seeks relief. Vesical symptoms are usually due to secondary cystitis, but acute urinary retention due to prolapse of the cyst into the urethra may occur.

*Incidence.* Ureterocele occurs most commonly in women, usually on the left side.

Bilateral involvement is seen in 10 to 15 per cent of cases. Age is not a factor in its development, for cases have been reported in the stillborn as well as in patients in their seventh decade. In most instances, however, the patient is from 30 to 35 years of age. A detailed urologic history will point to ureteral obstruction earlier in life, and with recent developments in pediatric urology, many more of these anomalies will be diagnosed in infants and children. Campbell reports encountering ureterocele in nineteen children in a group of 580 with persistent pyuria.

*Etiology.* There is some divergence of opinion as to the cause of ureterocele, but the almost universal finding of stenosis of the ureteral orifice suggests this as a major contributing factor. There is little to substantiate Petillo's belief that the essential factor is paralysis and subsequent atony with dilatation of the terminal ureter. The theory of congenital ureteral obstruction is supported most ably by Chwalle and Brown. In the embryo, the ureter shifts its attachment from the Wolffian duct to the bladder. Chwalle has shown that in all human embryos of 12 to 28 mm. there is a brief period of atresia of the ureteral lumen at the bladder well following the establishment of this new connection. The epithelium overlying the lumen then becomes thinner, bulges toward the bladder and finally ruptures, establishing patency of the ureter. Vermooten points out that if the pressure on the membrane does not cause it to slough and allow the escape of the fluid as a result of the active functioning of the metanephros, we must assume that a hydronephrosis and hydroureter will develop. That this hydronephrosis does develop is shown by Brown, who found a high percentage of mouse embryos, in a strain of mice, to have deformed or abnormal kidneys. She found that the hydronephrosis was produced by a developmental stricture of one or both ureters at their junction with the bladder. The condition was first noted at the seventeen day stage. In ten of a group of twelve embryos, the pelvis and the upper fourth of the ureter were greatly distended by fluid. The ureters, however, narrowed down to their junction with the bladder, where the muscular wall of the bladder appeared to form a stricture. Among the nineteen and twenty day embryos, she found three cases of definite hydronephrosis due to stricture of the ureter just before it entered the bladder. Vermooten points out that if the membrane occluding the ureter only partially breaks down, either a congenital valve, a ureterocele, or a stricture at or just above the ureterovesical junction develops.

These facts support the congenital theory of ureterocele formation, which is further emphasized by the fact that definite ureteroceles have been demonstrated in the newborn and occur frequently in association with other developmental anomalies. There is, however, no reason to believe that stricture or stenosis of the ureteral meatus may not be acquired in some instances as a result of inflammation.

*Diagnosis.* There are no pathognomonic signs or symptoms of ureterocele. The symptoms and findings may suggest pyelonephritis, stone or tumor. Diagnosis is dependent upon cystoscopy, although the characteristic appearance of a dilated lower ureter protruding into the bladder, noted in excretory urograms, may be highly suggestive. Early investigation of recurrent or chronic persistent pyuria in infants and children will allow many more of these lesions to be treated early, while there is less secondary renal and ureteral pathology. Cystoscopic examination in early cases shows a small pea-size cystic dilatation with a small orifice on its surface representing the stenosed ureteral orifice. In larger lesions, the orifice may be displaced in any direction. The cyst wall appears thin and translucent with well-defined vessels, although when infection is present, or in the rare, thick-walled cyst containing fibrous and muscle tissue between the two mucous layers, the wall appears thick and opaque. While observing the ureterocele through the cystoscope, one sees with each peristal-

tic rush of urine, a ballooning-up of the cyst followed by efflux from the stenosed orifice and collapse of the cyst. Frequently the ureteral orifice cannot be entered with anything but the smallest catheter. Search should be made for more than one orifice, for many cases of ureterocele are accompanied by reduplication of the ureter on the corresponding side. Retrograde or excretory urograms and renal function tests are important in estimating the condition of the upper urinary tract.

*Treatment.* Treatment depends upon the stage of evolution of the ureterocele, the presence or absence of complications, and the degree of renal and ureteral damage. Types of treatment are divided into surgical and urologic.

All treatment directed to the ureterocele per se is concerned with two objectives: first, to remove the obstruction at the terminal ureter; and second, to preserve the integrity of the ureterovesical valve. Many reports of otherwise successful operations, particularly those involving open surgery with resection of the ureterocele, state that the patient has been left with ureteral reflux, sometimes necessitating nephrectomy later. It is hardly necessary to point out that nothing can be accomplished in restoring competency of the ureteral valve by any transurethral procedure. Therefore, it would seem of immediate importance in treating ureterocele to estimate competency of the ureteral valve by all available preoperative means.

If the valve is incompetent, the procedure of choice is either cystotomy with removal of the cyst and repair of the aperture in the bladder wall in an attempt to restore ureteral valve competency, or immediate nephroureterectomy. It is beyond the scope of this paper to deal with these phases of the subject. Nephroureterectomy has been adequately described by Gibson. Young's operative procedure of bringing together the musculature of the bladder with a purse-string suture to repair the bladder wall, reported by him in 1912, was among the first of such reports. However, it is probably safe to say that when obstruction has been of such persistence and long standing that the intramural portion of the ureter is grossly dilated, the ureter and kidney will also be severely damaged, and it is wiser to elect nephroureterectomy at the start.

I agree with Petillo and Hunner who say ablation through suprapubic cystotomy, still advocated by many surgeons, is to be considered a surgical abuse, unless for special reasons it should be absolutely necessary. As an exception to this rule, one might consider the rare case of well advanced ureterocele with incompetent ureterovesical valve where sufficient good functioning renal tissue is present to warrant its conservation. Other exceptions would be the very large ureteroceles filling the bladder and causing urinary retention, or those extruding through the urethral orifice as reported by Kuth, Herick and Davis and Owens, in which cases the suprapubic approach might be indicated.

When the ureterocele is very small and almost insignificant in size, dilatation of the meatus with ureteral bougies and catheters, and irrigations of the renal pelvis and ureter at ten-day intervals are frequently sufficient to establish and maintain good drainage.

In the development of the larger ureteroceles, it must be kept in mind that as a result of increased ureteral pressure due to meatal obstruction, dilatation of the lower portion of the intramural segment of the ureter occurs. As it progresses in size, it strips the mucosa up from the musculature of the bladder but does not involve it with its sphincter-like action until later. Therefore, competency of the ureterovesical valve remains. This is better understood when one considers Gruber's illuminating work, showing that three-fourths of a dog's ureteral valve may be completely cut away without signs of regurgitation, even under extreme pressure. In defending the procedure of ureteral meatotomy in general, Moore reports its use favorably in cases of ureterocele. He states that the complication

most to be feared following meatotomy is regurgitation of urine from the bladder up the ureter, and reports this as occurring

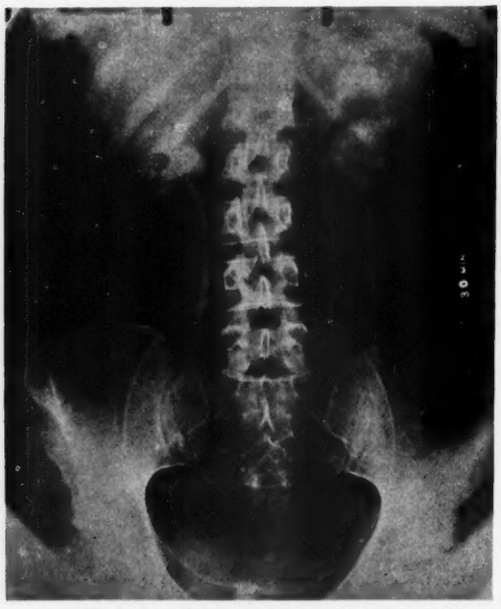


FIG. 1. Preoperative excretory urogram, showing double left renal pelvis and ureter, and moderate pyelectasis and ureterectasis. Good excretion of the contrast medium evident.

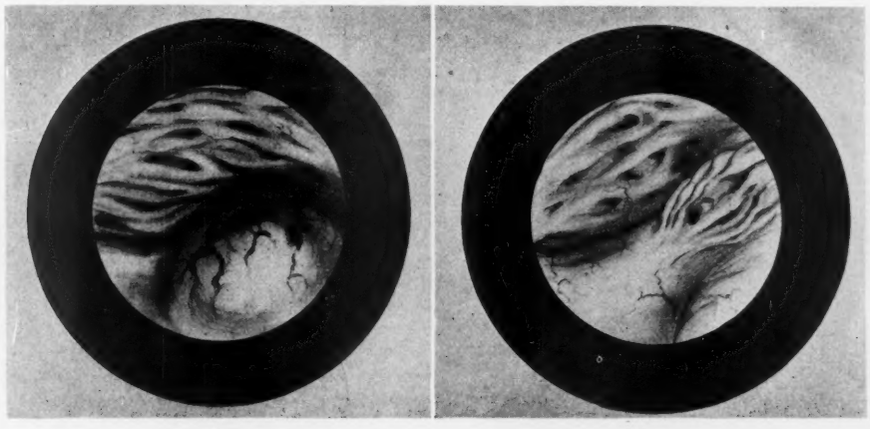
rarely in his experience. It would seem that if treatment is undertaken in the early stages of ureterocele development, before much dilatation of the intramural portion of the ureter has occurred, simple opening of the cyst, either at the meatus or elsewhere, should suffice to eliminate obstruction of the stenosed ureteral orifice, and should permit the ureter to drain freely into the bladder through a competent ureterovesical valve.

It is only of academic interest to consider the various methods with which transurethral opening of the ureterocele may be accomplished. Any method which establishes a good opening should suffice. The meatotomy can be accomplished with the cold cutting scissors or with the high frequency cutting current applied by an electrode. Special types of electrodes have been devised primarily for this purpose by Moore and Riba. If the ureteral orifice is difficult to see or enter, the cyst may be opened elsewhere.

Removal of the redundant portion of the cyst wall seems unnecessary although partial removal in the very large ureteroceles might be of advantage. Various authors report attacking ureteroceles through the cystoscope with the cautery loop, scissors, a small knife, cystoscopic rongeur, a combination of knife and punch force, or by fulguration. Riba points out that if the ureterocele is of a non-collapsible type, transurethral resection with the McCarthy electrotome is the treatment of choice and has used it in two of his cases. Gutierrez mentions the loop electrode method of treatment for endoscopic prostatic resection but has used it in none of his eighteen cases. These are the only references to this particular procedure in the literature of which I am aware.

*Procedure.* It would seem of interest to point out that most ureteroceles may be adequately dealt with by resection of a portion of the wall with the McCarthy electrotome which is now in such common usage. All but the very small and the very large ureteroceles are susceptible. A general anesthetic is necessary, preferably low spinal or intravenous. High intravesical pressure when the bladder is overdistended prevents ballooning of the ureterocele, so that a little or moderate distention after introduction of the instrument allows better visualization of the ureterocele. If the ureterocele is of the uncommon, thick-walled, non-collapsible variety, no difficulty will be experienced in resecting a large piece of tissue with the loop electrode. Some of the redundant wall may be resected if thought desirable. Fulguration adequately controls any bleeding which occurs. In the thin-walled, collapsible ureterocele, maximum ballooning must be watched for and at the precise moment it occurs resection should be carried out. This is followed immediately by collapse of the cyst and retraction of its wall; further resection of the wall is dangerous and unnecessary. The site selected for resection should be the most prominent portion of the cyst. If the ureterocele is sufficiently distended, there is

no danger of injuring or penetrating the musculature of the bladder beyond the bladder and ureteral mucosa comprising the cyst wall. The muscular bladder wall and intramural portion of the ureter are left intact. If the ureterovesical valve is competent, reflex will not occur. The redundant ureterocele wall shrinks, the raw surface of the bladder epithelializes and good ultimate repair occurs. Insertion of a retention ureteral catheter prevents possible ureteral obstruction incident to postoperative edema and a retention urethral catheter obviates possible harmful bladder distention. The ureteral catheter should be removed in two to four days, while the urethral catheter may be left for a longer time if desirable.



A B

Fig. 2. Cystoscopic appearance of ureterocele. A, ureterocele distended; B, ureterocele collapsed.

## CASE REPORT

Mrs. L. C., age 29, was admitted to the medical department of the University Hospital August 16, 1938. She complained of nausea, vomiting, chills, fever and dysuria. Two weeks prior to admission, the patient had "septic sore throat" lasting four days, after which she was well for two days and then had nausea and vomiting and, later, chills and fever. Burning on urination had been severe for five days before admission. Suprapublic and low back pain occurred one day before admission. The patient had had a normal child two months prior to admission. No part of the history suggested previous urinary infection or pathology.

Examination disclosed a reddened pharynx, some tenderness across the entire lower abdomen and slight tenderness in each kidney region, more noticeable on the left side. The urine was loaded with pus cells and contained many red blood cells. Flexner dysentery bacilli were cultured from the urine. Agglutinations for dysentery were positive in dilution 1:320.

Sulfanilamide in large doses caused rapid clearing of the urine and a drop in temperature from 105 on admission to normal five days later. The drug, however, caused marked cyanosis, the hemoglobin dropped to 65 per cent, and the erythrocytes to 3,230,000 six days after admission. Therefore, it was discontinued. On August 24, a transfusion of 375 c.c. of citrated blood was given. The patient did well until September 4, when the temperature suddenly rose from normal to 105, accompanied by a chill. She could not tolerate sulfanilamide at this time,

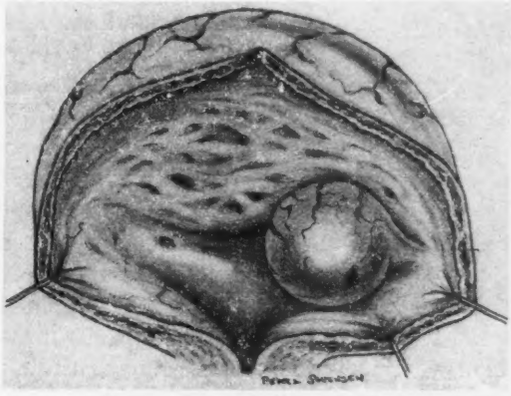


Fig. 3. Gross appearance of left ureterocele. Only one ureteral orifice seen.

although it was tried for a few days, with no appreciable effect on the pyuria and fever.

The excretory urograms made on September 1, (Fig. 1) showed a slightly enlarged left kidney, double left ureter and renal pelvis, and moderate pyelectasis and ureterectasis. There was good excretion of the contrast media by each kidney.

FIG. 4. Preoperative retrograde pyeloureterogram showing marked dilatation of the inferior left renal pelvis, and associated ureter down to its junction with the bladder. The upper pelvis has not been filled with contrast medium. Ureterocele not demonstrated.

Urologic consultation was requested on September 7. There was marked tenderness in the left costovertebral area but the left kidney was not palpably enlarged. Observation cystoscopy on September 8 showed a normal right ureteral orifice and a left ureterocele, which alternately filled (Fig. 2A) and collapsed (Fig. 2B) in association with ureteral peristalsis. When full, the cyst was about the size of an English walnut. (Fig. 3.) Only one ureteral orifice was seen on the left side. A No. 6 catheter was passed easily, relieving the left flank pain instantly; 75 c.c. of hazy urine was aspirated from the left renal pelvis.

Phenolsulphonephthalein appeared in six minutes from each ureteral catheter after intramuscular injection and 5 per cent was recovered in ten minutes from the right side, none from the left. Bilateral pyeloureterograms were obtained by injecting 30 c.c. of contrast media on the left and 5 c.c. on the right side. A No. 6 left retention catheter drained well for a period of 4 days, during which time the temperature rapidly subsided to normal and the pain in the left flank was relieved. Chills and fever were again noted on September 21, at which time another retention left ureteral catheter was inserted and again pain and fever rapidly subsided. No further urinary antiseptics were given, but an autogenous vaccine made from cultures of the urine was given from September 10 to September 18.

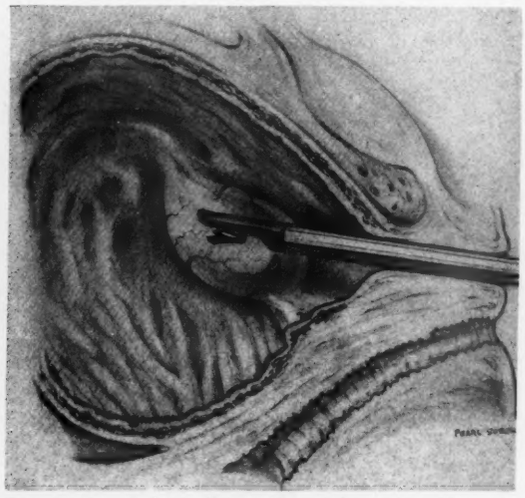


FIG. 5. Removing a large piece of ureterocele wall with the McCarthy electrotome. Site elected for resection is located over the most prominent portion of the ureterocele at a time when the cyst is fully distended.

The retrograde pyelograms (Fig. 4) showed marked dilatation of the inferior left renal pelvis and the associated left ureter down to its junction with the bladder. The ureterocele was not demonstrated.

On September 24, under spinal anesthesia (50 mg. novocaine), one large piece of tissue was removed from the most prominent portion of the ureterocele with the McCarthy resectoscope. (Fig. 5.) This tissue was removed at the time the ureterocele was seen to be fully distended. The ureterocele collapsed immediately. A No. 6 ureteral catheter was passed through this new opening into the left ureter where it was left for continuous drainage.

No postoperative reaction occurred. There was no elevation in temperature and the patient was entirely relieved of all left renal pain. The retention ureteral catheter was removed on the third postoperative day. The urine was clear on numerous examinations during her postoperative stay and the cultures were sterile. The

patient was transferred from the urologic service on October 2, the eighth postoperative day, to the psychiatric department because of toxic psychosis. Her mental condition rapidly improved and she was dismissed from the hospital, well, on October 16, 1938.

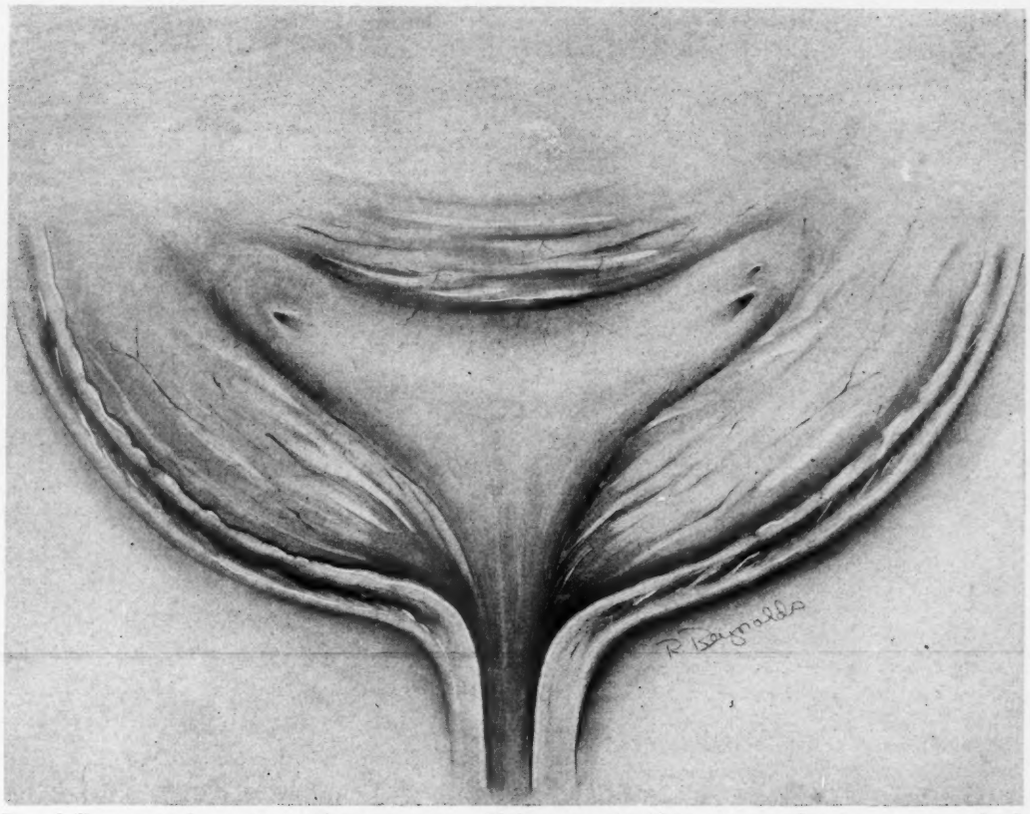


FIG. 6. Postoperative cystoscopic appearance of left ureteral orifices ten months after transurethral resection. Two orifices were seen, the upper slightly smaller and the lower slightly larger than normal.

The pathologic examination of the tissue removed at operation showed it to be covered with transitional epithelium. There were some strands of smooth muscle fibers in the wall.

This patient returned to the University Hospital on August 3, 1939, because of headaches and indefinite gastrointestinal symptoms. She complained also of occasional pain in the left flank when her bladder was overdistended. This suggested possible ureteral reflux. The left kidney was not tender or palpable. Cultures of the bladder urine showed *B. coli communis* and microscopic examination showed fifteen to twenty pus cells per high power field. The nonprotein nitrogen was 33.0 mg. per cent.

Observation cystoscopy showed two left ureteral orifices at this time, the lower one being slightly larger and the upper slightly smaller than normal. (Fig. 6.) Each orifice admitted a No. 6 ureteral catheter readily, the lower one passing to the upper pelvis, as would be expected. Urine from each left catheter showed appearance of phenolsulphonephthalein in five minutes after intravenous injection; 2.5 per cent was recovered from the upper and 1 per cent from the lower pelvis in ten minutes after appearance of the dye. Microscopic examination of the urine from the upper pelvis showed only a few pus cells and from the lower only five to eight pus cells. Culture of the urine from the upper pelvis was negative and from the lower showed *alkalinis aerogenes*. The retrograde pyelograms at this time (Fig. 7) indicated regression in size of the two left renal pelves and ureters although there was still some pyelectasis and ureterectasis. The cystogram indicated no *x*-ray evidence of ureteral reflux. The patient was discharged from the hospital after two weeks' observation, entirely free of symptoms.

## COMMENT

Apparently this patient had no symptoms referable to the bladder or the left

upper urinary tract until a hematogenous urinary infection occurred incident to "septic sore throat." This seems to be the usual

Fig. 7. Postoperative retrograde pyeloureterogram ten months after transurethral resection, showing definite regression in size of the left ureters and renal pelvis indicating improved drainage although some pyelectasis and ureterectasis persists.

story, although as pointed out by Hunner, minor symptoms of ureteral obstruction should be elicited by careful history if the patient comes under observation prior to the onset of infection. The fact that an indwelling ureteral catheter caused prompt regression of symptoms emphasizes the importance of drainage in these cases. In spite of the well advanced hydroureter and hydronephrosis, accompanied by marked impairment of renal function, relief of obstruction caused regression of the dilatation and return of much function. Infection was practically eliminated, as were the patient's symptoms. In spite of apparent moderate dilatation of the lower ureter preoperatively, the ureterovesical valve remained competent as demonstrated postoperatively by absence of ureteral reflux in the cystogram. Simple transurethral resection of a portion of the ureterocele wall without removal of the redundant portion of the cyst caused no postoperative reaction, produced good ureteral drainage, and maintained competency of the ureterovesical valve. It seems that a large majority of ureteroceles would be amenable to such surgical attack. The appearance of the bladder one year following operation indicated excellent repair and good end result. The lower left orifice probably represented the new opening established by resection. The small upper left ureter readily admitted a No. 6 ureteral catheter. Reduplication of the ureters in this case further emphasizes the dictum that when one congenital anomaly of the urinary tract is encountered, others should be suspected.

## SUMMARY

1. The most probable developmental cause of ureterocele is discussed.

2. Early recognition of ureterocele by complete urologic investigation of recurrent or persistent pyuria in children is emphasized.

3. Various methods of treatment of ureteroceles are considered and the suggestion is made that many are amenable to simple transurethral resection of a portion of the wall with an electrotome.

4. One case of ureterocele, with reduplication of the ureter and pelvis, treated by transurethral resection is reported.*

## REFERENCES

1. Brown, A. L. An analysis of the developing metanephros in mouse embryos with abnormal kidneys. *Am. J. Anat.*, 47: 117, 1931.
2. Campbell, M. F. Surgical treatment of anomalies of the upper urinary tract in children. *J. A. M. A.*, 106: 193, 1938.
3. Chwalle, R. Process of formation of cystic dilatations of the vesical end of the ureter, etc. *Urol. & Cutan. Rev.*, 31: 499, 1927.
4. Davis, E. and Owens, C. A. Prolapse of the ureter through the urethra. *Tr. A. A. Genito-Urin. Surg.*, 21: 367, 1928.
5. Dawley, W. A. Ureterocele. *Arch. Surg.*, 21: 458, 1930.

* Since this paper was submitted for publication, the author has treated an additional case of bilateral ureterocele in a 60 year old female patient by the transurethral method herein described, with similar early relief from symptoms, although not enough time has elapsed for final opinion as to late results.

6. FOLEY, F. E. B. New method for cystoscopic surgical treatment of ureterocele. *Urol. & Cutan. Rev.*, 35: 49, 1931.
7. GIBSON, T. E. A new technique of nephro-ureterectomy with special reference to ureterocele. *Am. J. Surg.*, 34: 206, 1936.
8. GRUBER, C. M. The function of the uretero-vesical valve and the experimental production of hydroureters without obstruction. *J. Urol.*, 23: 161, 1930.
9. GUTIERREZ, R. Modern surgical treatment of ureterocele. *Surg., Gynec. & Obst.*, 68: 611, 1939.
10. HERRICK, F. C. Large ureterocele with herniation through the urethra. *J. Urol.*, 35: 643, 1936.
11. HUNNER, G. L. Ureterocele. Report of 10 cases. *Urol. & Cutan. Rev.*, 39: 755, 1935.
12. KUTH, P. E. A large ureterocele which extruded from female bladder on voiding. *J. Urol.*, 42: 534, 1939.
13. LAZARUS, J. A. Cystic dilatation of the lower end of the ureter. Special reference to the transurethral treatment with the high frequency cutting current; report of two cases. *J. Urol.*, 36: 139, 1936.
14. MCKENNA, W. R. Ureterocele. *S. Clin. North America*, 7: 1001, 1927.
15. MOORE, N. S. Ureteral meatotomy; a clinical evaluation. *J. Urol.*, 26: 519, 1931.
16. O'CONOR, V. J., and JOHNSON, A. B. Ureterocele. A clinical study of nineteen cases. *J. Urol.*, 23: 33, 1930.
17. PATCH, F. S. Ureterocele, with report of a case. *J. Urol.*, 16: 125, 1926.
18. PETILLO, D. Ureterocele: clinical significance and process of formation. Report of four cases. *Surg., Gynec. & Obst.*, 40: 811, 1925.
19. RHODES, J. S. The clinical importance of ureterocele. *J. Urol.*, 35: 300, 1936.
20. RIBA, W. L. Ureterocele: with case reports of bilateral ureterocele in identical twins. *Brit. J. Urol.*, 8: 119, 1936.
21. VERMOOTEN, V. A new etiology for certain types of the dilated ureters in children. *J. Urol.*, 41: 455, 1939.
22. YOUNG, H. H. Report of a case of double ureter with intravesical ballooning of its lower ends. *Tr. Am. Urol. A.*, April 3, 1912.

# TREATMENT OF FURUNCLES, CARBUNCLES AND ABSCESSES OF STAPHYLOCOCCIC ORIGIN WITH THIAZOLE DERIVATIVES OF SULFANILAMIDE

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SINCE Gelmo[5] synthesized para-amino-benzene-sulfonamide in 1908, numerous variations have been developed. None have been both effective against staphylococci and innocuous to the host. This has also been true of other compounds not related to sulfanilamide or to the azo dyes. In 1939, a new series of derivatives was announced by Fosbinder and Walter[4] and subsequently by Lott and Bergeim.[9] Among them were 2 (para-amino-benzene-sulfonamido) thiazole and its methylated derivative, 2 (para-amino-benzene-sulfonamido) 4-methylthiazole. They are more commonly known by the names sulfathiazole and sulfamethylthiazole, respectively.*

Preliminary studies[1,6,10,12] on animals and on humans seemed to indicate that the thiazoles were not more toxic than sulfapyridine and had a more profound effect against staphylococci. Since sulfamethylthiazole appeared to be the most potent in this respect, one of us (C.A.B) undertook an investigation of the use of the drug for acute staphylococcic infections that are frequently fatal. During the investigation the collaboration of the coauthor (A. R. A.) resulted in this paper concerning the less severe staphylococcic infections.

The initial study showed that sulfamethylthiazole was more effective against staphylococci in vivo than sulfapyridine, and that the reactions produced by the former were fewer and less severe. The drug was given, therefore, to hospitalized patients with milder staphylococcic infections, including furuncles and abscesses. Our experience with sulfamethylthiazole was encouraging and indicated that it was not a dangerous drug in nearly fifty cases of various types to which it was administered in various forms. Other investigators observed peripheral neuritis in several cases[7] and the condition was confined almost exclusively to ambulatory patients. Since no neuritis has been reported in patients with pneumococcal pneumonia treated with sulfathiazole[3] we considered that it was safe to use sulfathiazole for ambulatory patients with furunculosis.

This communication is limited to forty cases of staphylococcic infections. They were not selected, but were taken as they were admitted for hospitalization or presented themselves in surgical clinics for treatment. There were thirteen patients with carbuncles, twelve with furuncles, and fifteen with abscesses. Twenty-five patients received sulfamethylthiazole and the remaining fifteen sulfathiazole. Staphylococci were cultured from the lesions in each patient and were recovered from the blood stream of one. All determinations of the level of the drugs in the blood, urine and feces were done according to the method of Bratton and Marshall.[2]

## ADMINISTRATION AND DOSAGE

The methods of administration and the dosage were the same for both drugs. Half-gram (7½ grain) tablets were given whole or powdered and suspended in milk or condensed-milk and milk in about equal proportions. These seemed to be the most satisfactory when a suspending medium was required. The earlier patients received sulfamethylthiazole in powdered form made up into ½ Gm. capsules. The first few

* The sulfamethylthiazole and sulfathiazole were supplied to us by the Department of Medical Research of the Winthrop Chemical Company.

adults were given an initial dose of 3 Gm. followed by 2 Gm. every four hours day and night. We found that this dose was larger than necessary and reduced it to 1 Gm. every four hours. The level of free drug in the blood reaches as high as 11 mg. per cent when the larger dose is used, with an average around 8 mg. per cent. With the 1 Gm. dose the concentration of free drug is usually around 4 mg. per cent and any concentration above 3 mg. per cent seems to be adequate for the types of cases herein reported. If the dose is reduced to below 1 Gm. every four hours, much of the effect is lost. We now give the average adult an initial dose of 2 Gm. followed by 1 Gm. every four hours, omitting the 4 A.M. medication after forty-eight hours. A total of 5 to 6 Gm. is received daily (75 to 90 grains). Children are given 0.5 Gm. per twenty-five pounds of body weight to start with and a maintenance dose of 0.25 Gm. per twenty-five pounds every four hours, both calculated to the nearest ½ Gm. It may often be necessary to increase the dosage to maintain a free drug blood level that is at all satisfactory. Children apparently eliminate the thiazoles rapidly and tolerate them very well.

Two patients were given rectal instillations of the drug. One received sulfamethylthiazole and the other, sulfathiazole. The first required rectal administration because of extreme nausea and vomiting. She maintained a blood level of at least 0.15 mg. per cent while taking 2 Gm. every four hours by rectum. The other patient received sulfathiazole by rectum for two days because she refused to take it by mouth after experiencing slight nausea. She tired of the rectal feedings and returned to the usual methods after two days without further nausea. None of the patients were sufficiently ill to require immediate creation of an adequate blood level by giving the sodium salts intravenously.

### ABSORPTION AND EXCRETION

Absorption of the thiazoles from the human gastrointestinal tract must depend upon a number of factors which include the rapidity of passage of the material through the tract and the condition of the mucous membranes of the various portions of the tract with regard to their power of absorption at the time. Individual variations in blood level obtained from the same oral dosages demonstrate this. The absorption rate must, therefore, be calculated from the sodium salts which are more uniformly absorbed. This is not practical for clinical purposes. As stated in the preceding section, an initial dose of 2 Gm. followed by 1 Gm. every four hours will usually result in a blood level of free drug in adults of about 4.0 mg. per cent, and almost always in a level above 2.9 mg. per cent. Absorption continues with larger doses and it is possible to cause a rise in blood level to over 20 mg. per cent.

Sulfathiazole and sulfamethylthiazole exist in the blood stream in free and conjugated or acetylated forms. The amount of conjugated sulfathiazole or sulfamethylthiazole is usually low in relation to the free drug. The proportion of conjugated drug is relatively high in the urine and very low in the stools. Analysis of the tables compiled by Reinhold, Flippin and Schwartz[11] from patients with pneumonia given repeated doses of sulfathiazole reveals that 25 per cent of the total drug in the blood is in conjugated form, taken as a grand average with maximum deviations to 2 per cent and 70 per cent. The average ratio of sulfathiazole to acetylsulfathiazole is, therefore, three to one. It is a fairly constant ratio except for occasional wider variations.

The blood stream is entirely cleared of the drug, as estimated by the routine tests, in twenty-four to forty-eight hours depending upon the amount of drug in the circulation at the time it is discontinued. Excretion takes place through the kidneys and gastrointestinal tract so that the two forms of the drug may be recovered from the urine and feces. The following table shows the proportion of a 3 Gm. oral dose excreted in twenty-four hours through the several channels.

### TOLERATION AND REACTIONS

The chemical nature of the thiazoles and their relationship to the parent sulfanilamide and to sulfapyridine and other derivatives would suggest that side effects might be produced that are similar to those from other compounds used in humans. We were constantly on the watch, therefore, for the appearance of such reactions as anemia, leukopenia, cyanosis, nausea, vomiting, diarrhea, skin rashes, hyperpyrexia, nephritis, hematuria, urinary lithiasis and psychic disturbances.

WHITE MALE, 41 YEARS, 150 POUNDS

| Date | Hour | Blood | Urine | Feces | Combined Excretion | Form |
|---|---|---|---|---|---|---|
| July, 31, 1940 | 9 A.M. | 3 Gm.—single oral dose | | | | |
| | 3 P.M. | 1.8 mg.% | .... | .... | ..... | Free |
| | 3 P.M. | 2.3 mg.% | .... | .... | ..... | Total |
| August 1, 1940 | 9 A.M. | 0.1 mg.% | 0.1 Gm. | 0.24 | ..... | Free |
| | 9 A.M. | 0.2 mg.% | 2.36 | 0.27 | 2.57 | Total |
| August 2, 1940 | 9 A.M. | 0 mg.% | 0.0 | 0.05 | ..... | Free |
| | 9 A.M. | 0 mg.% | 0.2 | 0.05 | 0.25 | Total |
| July 31–August 2, 1940 | Total drug recovered | | | | 2.82 Gm. | |

REACTIONS TO DRUGS

| Reaction | Patients Given Sulfamethylthiazole (25) | | Patients Given Sulfathiazole (15) | |
|---|---|---|---|---|
| | No. | Per Cent | No. | Per Cent |
| Nausea | 9 | 35 | 5 | 33 |
| Vomiting | 3 | 12 | 2 | 12 |
| Crystals in urine | 16 | 65 | 1 | 6 |
| Headache | 3 | 12 | 1 | 6 |
| Hematuria (micro.) | 2 | 8 | 2 | 12 |
| Skin rash | 2 | 8 | 0 | |
| Weakness | 1 | 4 | 1 | 6 |
| Anemia | 2 | 8 | 0 | |

Because a small series limits justifiable conclusions, the various reactions encountered are listed in their order of frequency and no attempt has been made to set up a statistical analysis.

The fact that no patients receiving either of the thiazoles have exhibited reduction in the number of leukocytes either during or after treatment merits special emphasis, particularly in view of the absence of such reaction in the prior series of grave staphylococcic infections. Two patients that received sulfamethylthiazole evidenced a moderate anemia which rapidly cleared up after discontinuing medication. The appearance of anemia was not considered an indication for withdrawal. Two other patients taking sulfamethylthiazole developed diffuse punctate erythematous rashes on the body and extremities. These rashes disappeared within twenty-four to forty-eight hours although medication was continued.

The majority of patients do not have gastric distress. Others complain of vague discomfort and a loss of desire for food. When nausea occurs, it is usually not accompanied by vomiting and disappears promptly while the dosage is maintained. The development of nausea does not seem to be related to the occurrence of other reactions. It comes on more often when sulfamethylthiazole is given. The 33 per cent incidence of nausea recorded for sulfathiazole is not representative because of the few cases and it has been our experience with a larger series of over fifty patients treated with this drug that nausea is less frequent. One woman who received sulfamethylthiazole was violently nauseated and vomited persistently until it was discontinued. She tolerated it by rectum. Large numbers of crystals were found in the

urine from most of the patients receiving sulfamethylthiazole, while the urine of those taking sulfathiazole rarely contained

that the urea clearance was diminished as much as 50 per cent during sulfathiazole therapy, that it was transitory and that

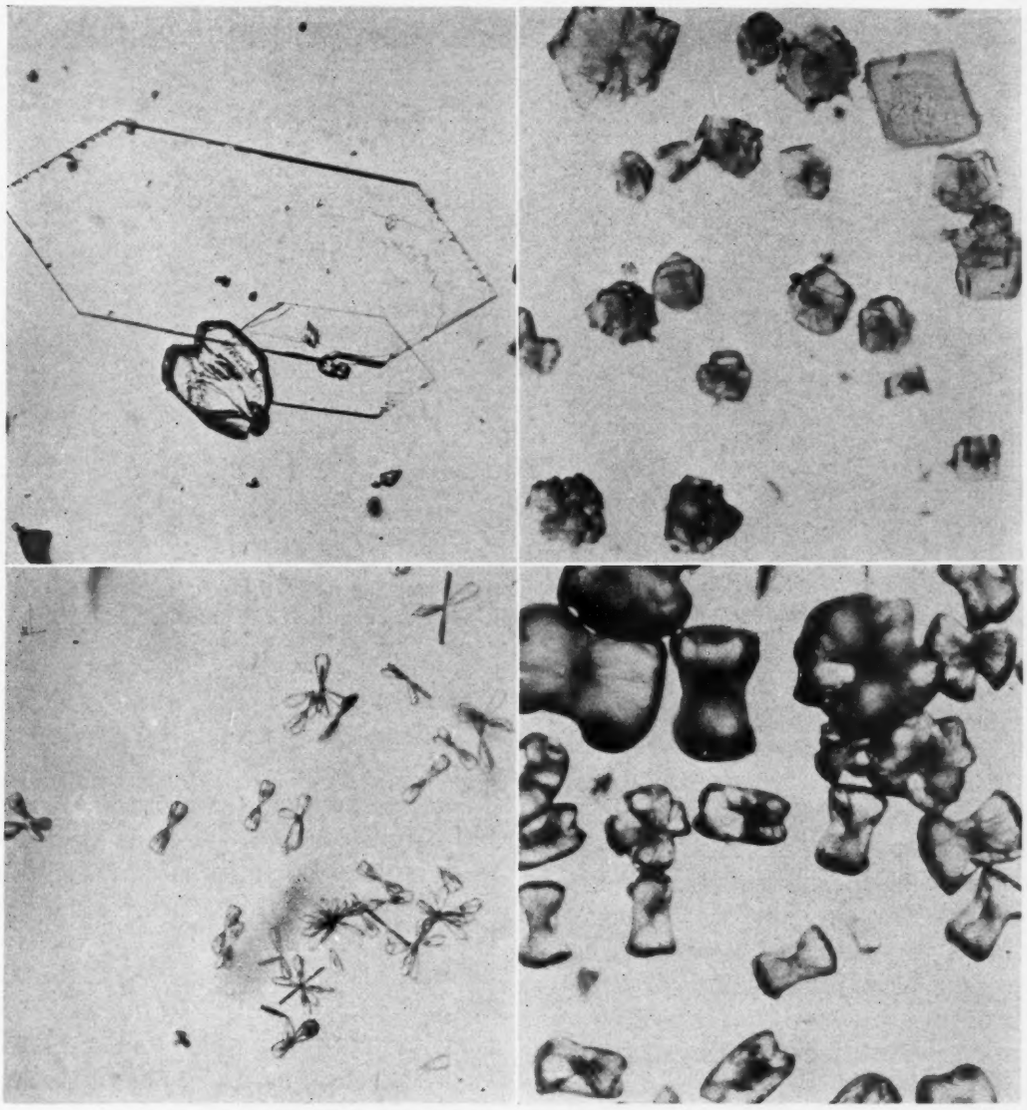

FIG. 1. Upper left: Sulfathiazole crystallized from neutral distilled water. Lower left: Sulfamethylthiazole in urine of treated patient. Upper right: Sulfathiazole dissolved and recrystallized from normal urine. Lower right: Sulfathiazole in urine of treated patient.

them. Red cells and crystals disappeared after medication was discontinued.

The low incidence of reactions of any consequence make sulfathiazole a relatively safe drug for use in ordinary staphylococcic infections. Aside from crystals and slight microscopic hematuria, no other changes in the urine were found. Diminished daily output was not observed. The findings of Reinhold, Flippin and Schwartz[11] in this direction merit attention. They observed return to normal function occurred without discontinuing the drug. The presence of pneumonia in all the cases and other factors probably enter into the final analysis.

Figure 1 depicts crystals of sulfamethylthiazole and sulfathiazole in the urine. The former are easily mistaken by the laboratory technician for urates and phosphates, and the latter for leucine and tyrosine.

Our patients have tolerated sulfamethylthiazole and sulfathiazole in oral doses of

from 6 to 12 Gm. daily for periods of from one to two and one-half weeks without any apparent cumulative toxic effect.

Fig. 2. O. M. H., No. 56082. Large furuncle of nose. Resolution following spontaneous drainage during three days of sulfamethylthiazole therapy. No drug reaction; 24 Gm. total; blood level, 5.0 mg. per cent.

### EFFECTS OF TREATMENT

Thirty-eight of the patients were definitely improved after receiving a considerable quantity of sulfamethylthiazole. One result was questionable because of the prolonged course of the disease and concomitant administration of staphylococcus vaccine. Staphylococci were cultured from every patient and in several cases streptococci were found.

Furuncles were affected most readily and disappeared within four days, except in two instances. One patient had about ten furuncles and responded satisfactorily to therapy although eight days elapsed before the last one disappeared. The other patient was not definitely improved and had received vaccine, as mentioned above. The most rapid disappearance of a furuncle occurred in two and one-half days, during which time a large, fiery-red indurated lesion on the nose receded to an almost indistinguishable tinge of redness without induration or edema. (Fig. 2.) It may be said that the length of time taken to respond to thiazole therapy will depend in part upon the stage of development of the lesion when the drug is commenced. We have observed that the earlier furuncles respond about 25 per cent more slowly than well developed lesions, but recede without pus formation. The latter quickly soften, discharge their contents promptly and heal. No core forms and the discharge is liquid, quite unlike that of the untreated furuncle. The induration and redness around the lesion disappear rapidly. The relative lack of induration is the most striking feature. After four days of sulfamethylthiazole therapy the drainage from the lesion in Case 11 became sterile. The original cultures disclosed Staphylococcus albus.

Case 1. The patient, a white female, ambulatory, 26 years of age, weighing 136 pounds, was referred by her physician for treatment.

Four days previously, on June 10, she noticed a "deep pimple" on her left cheek. This enlarged and became swollen. The lesion measured 4 cm. in diameter, was red, warm to the touch and fluctuant. Temperature was 99.2°F. Commencing on June 14 she was given sulfathiazole, 1 Gm. every four hours, omitting one dose at 4 A.M. Within twenty-four hours the lesion had softened and had started to drain. The patient complained of nausea, headache and malaise but did not vomit. The blood level

of the free drug was 3.6 mg. per cent. Cultures revealed Staphylococcus aureus. On the second day profuse drainage was followed by gradual

Fig. 3. O. M. H., No. 55632, Case 19. Before and after six days of sulfamethylthiazole therapy. Carbuncle of face and diabetes mellitus. Reaction—skin rash. 61 Gm. total; blood level, 5.5 to 9 mg. per cent.

disappearance of redness and induration. The core separated and came away on the third day. Nausea and headache had disappeared. The dose was reduced to 0.5 Gm. every four hours on the third day and medication discontinued on the fifth day. At discharge from the hospital the temperature was normal, the lesion was healing and there was practically no drainage. The urine was normal. The blood count was HG. 80 per cent red blood count, 4,200,000; white blood count, 6,700; polymorphonuclears, 78 per cent; lymphocytes, 22 per cent. A total of 19 Gm. was given.

Carbuncles respond more slowly because of the extensive nature of the lesion, but do so with relative rapidity and in essentially the same manner. The surrounding infiltrated areas become soft and the redness disappears while the drainage assumes the character described for furuncles. Any cores which may have formed prior to the institution of treatment are very soft and easily dislodged. Diabetes mellitus is not a contraindication to administration of the thiazoles although it may prolong the time required for the lesion to improve. This is not always true as is demonstrated by Case 11, which responded in six days. If it is deemed necessary to excise a carbuncle we advise giving sulfathiazole for twenty-four hours prior to operation. If immediate surgery is contemplated, sufficient sodium sulfathiazole should be injected intravenously to assure an adequate blood level. We believe that this will tend to prevent the spread of infection from the original site, to prevent the development of septicemia and to insure against residual infection locally. The following two cases are notable for their rapid recovery from carbuncles of the dangerous area of the face. The second patient had a staphylococcus bacteremia.

Case 11. O. M. H. No. 55632. A white male, 68 years of age, weighing 182 pounds, had had diabetes for several years.

About two and one-half weeks before admission, he developed a slight swelling on his forehead above the left side of the nose. The lesion gradually increased to involve the eye and cheek of the same side. On admission, February 8, 1940, there was a large, bluish red swelling on the forehead, the eyelids were swollen and bluish and the right eye was entirely closed. The discoloration extended in a

butterfly pattern over the dorsum of the nose and both cheeks. A small amount of yellow pus exuded from several small openings just above

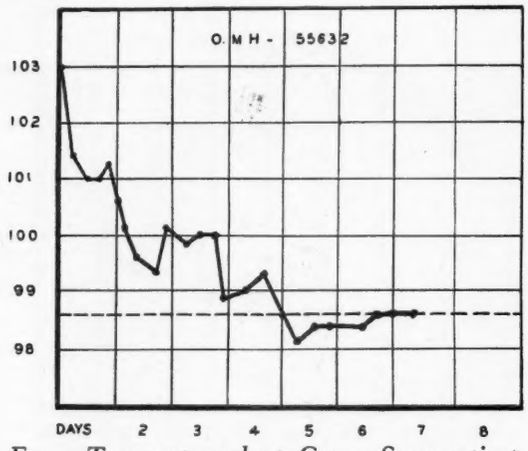


FIG. 4. Temperature chart, Case 2. Same patient as in Figure 3.

the inner canthus of the right eye. It flowed over the eyelid and appeared to be reaching the surface of the eyeball between the swollen lids. The temperature was 103°F., pulse 112, respirations 24. Cultures showed Staphylococcus albus and nonhemolytic streptococcus. The urine contained 4 plus sugar, 2 plus albumin, 4 plus acetone, and occasional hyaline casts. The blood culture was negative. The blood count was HG. 93 per cent; red blood count, 4,860,000; white blood count, 21,500; polymorphonuclears, 86 per cent. The blood sugar was 166 mg. He was given 2 Gm. every four hours night and day. Twenty-four hours later the temperature was 100.6°F., pulse 82, respirations 18. The urine contained 1 plus albumin, 20 to 25 red cells per HPF, and was free of sugar. The white count had dropped to 14,800 with 80 per cent polymorphonuclears. The carbuncle had softened considerably and was drained through a small incision at its most prominent point. Profuse drainage of pus followed. From this point on there was rapid improvement. Drainage was complete in four days. On the third day the temperature and pulse dropped to normal and remained there. The sulfamethylthiazole was reduced to 1 Gm. every four hours on the fifth day and was discontinued on the seventh day. On the eighth day the patient was transferred to the medical ward for diabetic treatment. The free sulfamethylthiazole content of the blood reached 5.5 mg. per cent in twenty-four hours. It gradually increased to 9 mg. per cent on the fifth day and when the dose was reduced it dropped to 6.6 mg. per cent. The blood count at the close of the treatment was HG. 91 per cent; red blood count, 4,560,000; white blood count, 8,000; polymorphonuclears, 66 per cent. Red cells were found in his urine on two occasions but disappeared about twenty-four hours after the medication was discontinued. He was not nauseated or cyanotic and did not vomit. On the fourth day a generalized reddish, blotchy skin rash developed that caused some itching, but disappeared in twenty-four hours without interruption in the administration of sulfamethylthiazole. A total of 61 Gm. was given. (Figs. 3 and 4.)

CASE III. St. B. No. 9723. The patient, a white male, 38 years of age, weighing 148 pounds, had had increasing tenderness, redness and swelling of left upper lip for two days followed by fever and intense pain. The lesion spread up the nose to the left eye. It was the third attack in a year, but the first with general symptoms. The lip was greatly swollen and the nose and lower lid were red and edematous. The temperature spiked from 99.0° to 102°F. Blood culture on first day of hospitalization showed Staphylococcus aureus hemolyticus. On the third day in hospital sulfamethylthiazole was given—3 Gm., followed by 2 Gm. every four hours day and night. The blood level of free drug ranged between 4.8 mg. per cent to 6.8 mg. per cent during the eleven days he took medication. The lesion rapidly localized and none of the subsequent blood cultures were positive. Seven days after commencing the drug the lesion was localized to a small, red area in the left upper lip. On the twelfth day he complained of weakness in the shoulders and legs. This cleared up in about one week. His urine repeatedly contained many crystals of sulfamethylthiazole. The leukocytes declined from 13,500 to 7,900 in five days. Leukopenia or anemia did not occur. His hemoglobin, however, dropped from around 95 to 70 per cent, but returned to the former level in about one week after discontinuing the drug. A total of 119 Gm. was given. (Fig. 5.)

Abscesses usually respond to treatment in from five to eight days, depending upon the stage they have achieved before institution of treatment and upon the presence of cellulitis. If staphylococcic cellulitis is treated before there is any tendency to localization, resolution may take place

without pus formation. If pus does form, the abscess will be insignificant. When an abscess has already begun to form, the methylthiazole therapy was commenced on the sixteenth day and kept up for eight days, gradually tapering off at the end. A blood level

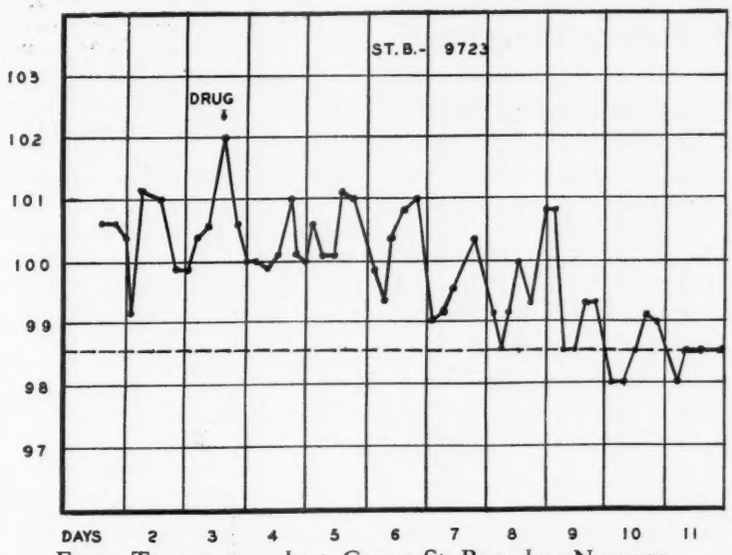


Fig. 5. Temperature chart, Case 3. St. Barnabas, No. 9723.

time taken to clear up the cellulitis and the abscess together is usually several days longer. One case with cellulitis and abscesses took twelve days, while Case IV, with multiple intractable abscesses throughout both breasts cleared up within five days. Another patient failed to be affected by sulfamethylthiazole. She had honeycombed abscesses throughout the scalp and subaponeurotic region following an injury to the scalp and extensive cellulitis. She also had diabetes.

Case IV. E. O. G. H., No. 38840. A white female, 38 years of age, weighing 120 pounds, was delivered of a child five weeks previously and subsequently discharged in good condition. Several weeks later she was admitted for incision and drainage of an abscess of the right breast and allowed to go home the following day. Four days later, during an attack of grippe, her left breast became painful and swollen. Fluctuation developed and she was again admitted for incision and drainage. The left breast was swollen, red, and painful to touch. Abscesses were honeycombed throughout the left breast and considerable fluctuation could be detected. Cultures from the abscesses revealed hemolytic staphylococcus aureus. They persisted for two weeks and were not improved by the usual hot, wet dressings, etc. Sulfa- of free drug of 3.1 mg. per cent was attained and she received 23 Gm. altogether. On the fourth day she complained of nausea which subsided after several subsequent doses and did not return. The case is of particular interest because of the great rapidity with which the previously intractable abscesses disappeared. There was no recurrence after her last discharge from the hospital. Urine and blood counts were entirely normal except for a leukocytosis at first, due to the infection.

## COMMENT

It must be understood that chemotherapy alone is not sufficient treatment for abscesses or any other collection of pus that is accessible. Incision and drainage are fully as important. Drugs and proper surgery should supplement each other and the former should also be employed to prevent spread of infection. If there is an abscess containing purulent material under tension, the patient's temperature will remain elevated despite continued thiazole therapy and will subside promptly following incision and drainage. The contents may be found to be sterile.

Furuncles should not be subjected to surgical interference. Such treatment is made all the more unnecessary by the

thiazoles. Hot, wet dressings of magnesium sulfate or boric acid should be employed as usual. In other words, these various lesions should be handled in the customary manner with the addition of thiazole medication by a suitable route.

While it is difficult to estimate accurately the value of drugs in terms of clinical improvement, certain reasonable criteria can be set up. If a furuncle, carbuncle or abscess treated with a drug disappears in less time than the shortest time required for an untreated lesion of the same kind, and if a sufficiently large number of these do the same thing, we are justified in accrediting the result to the particular type of therapy used, being careful to see that other medications and treatment do not influence the result.

The series herein presented fulfills these criteria, and, therefore, we claim that the thiazoles are of definite value in the treatment of staphylococcic lesions such as furuncles, carbuncles and abscesses.

## REFERENCES


1. Barlow, E. and Homburger, E. Specific chemotherapy of experimental staphylococcus infections with thiazole derivatives of sulfanilamide. *Proc. Soc. Exper. Biol. & Med.*, 42: 792, 1939.
2. Bratton, A. C. and Marshall, E. K., Jr. *J. Biol. Chem.*, 128: 537–550, 1939.
3. Flippin, H. F., Schwartz, L. and Rose, S. B. The comparative effectiveness and toxicity of sulfathiazole and sulfapyridine in pneumococcic pneumonia. *Ann. Int. Med.*, 13: 2038–2049, 1940.
4. Fosbinder, R. J. and Walter, L. A. Sulfanilamide derivatives of heterocyclic amines. *J. Am. Chem. Soc.*, 61: 2032, 1939.
5. Gelmo, P. Ueber Sulfamide der p-Amidobenzolsulfonsäure. *J. prakt. Chem.*, 77: 369, 1908.
6. Herrell, W. E. and Brown, A. E. The clinical use of sulfamethylthiazole in infections caused by Staphylococcus aureus. *Proc. Staff Meet., Mayo Clinic*, 14: 753, 1939.
7. Houghton, F. E. Personal communication.
8. Long, Perrin H. Thiazole derivatives of sulfanilamide. *J. A. M. A.*, 114: 870–871, 1940.
9. Lott, W. A. and Bergeim, F. H. Communications to the Editor: 2-(p-Aminobenzenesulfonamido)-thiazole: A new chemotherapeutic agent. *J. Am. Chem. Soc.*, 61: 3593, 1939.
10. McKee, C. M., Rake, G., Greep, R. O. and van Dyke, H. D. Therapeutic effect of sulfathiazole and sulfapyridine. *Proc. Soc. Exper. Biol. & Med.*, 42: 417, 1939.
11. Rheinhold, J. J., Flippin, H. F. and Schwartz, L. Observations on the pharmacology and toxicology of sulfathiazole in man. *Am. J. Med. Sc.*, 199: 393–401, 1940.
12. van Dyke, H. B., Greep, R. O., Rake, G. and McKee, C. M. Observations on the toxicology of sulfathiazole and sulfapyridine. *Proc. Soc. Exper. Biol. & Med.*, 42: 410, 1939.

# TWO POINT COAGULATION

## A NEW PRINCIPLE AND INSTRUMENT FOR APPLYING COAGULATION CURRENT IN NEUROSURGERY

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THE usual bipolar current to produce hemostasis in neurosurgery has done much to make present day neurosurgery possible. This method employs a common electrode applied to the patient's body which covers so large a surface that no local effect is produced at this electrode. At the active electrode the point which applies the current is so small that only a minute area of tissue is affected and this area is subject to considerable coagulation.

With a current strong enough to produce coagulation there is some damage to surrounding tissues since the current must spread by eventually passing to the common or body electrode. The farther from the active electrode, the greater the mass of tissue bearing the current and therefore the less damage to any molecular part. (Fig. 1.)

A current strong enough to produce the needed coagulation of bleeding vessels in scalp, dura or brain produces some damage to the surrounding tissue, with slight carbonization, necrosis and later tissue reaction. This is particularly undesirable in the brain itself, where the current may be carried along a vessel, resulting in tissue changes at a considerable distance from the point of coagulation. There is also a rather objectionable contraction of the vessel being coagulated which may shrink it beyond the field of vision even before complete hemostasis is secured. This shrinking is extremely noticeable in dural vessels. If coagulation is not carefully done, with minute amounts of current, later closure is difficult, and postoperative tissue reaction must ensue. These factors have limited the scope of electrocoagulation and have caused some authors, notably Bailey, to be exceedingly critical of its use.

In attempting to remedy some of the evils we have constantly kept a nurse at the electrocoagulation unit so that the coagulating current might be varied continuously to use only the smallest possible amount. The smaller the active electrode the smaller the amount of current necessary. The usual ball type electrode was therefore replaced by fine tipped forceps with a wire attached to the handle. (Fig. 2.) If a vessel had to be coagulated with a large current (occipital sinus for example) a hemostat placed between the coagulating forceps (or hemostat and grounded to the patient) could be used effectively to bypass the current so that the effect did not pass beyond this electrical short circuit into the vessel and deeper tissue. (Fig. 3.) This prevented shrinking of the vessel and obviously minimized tissue changes.

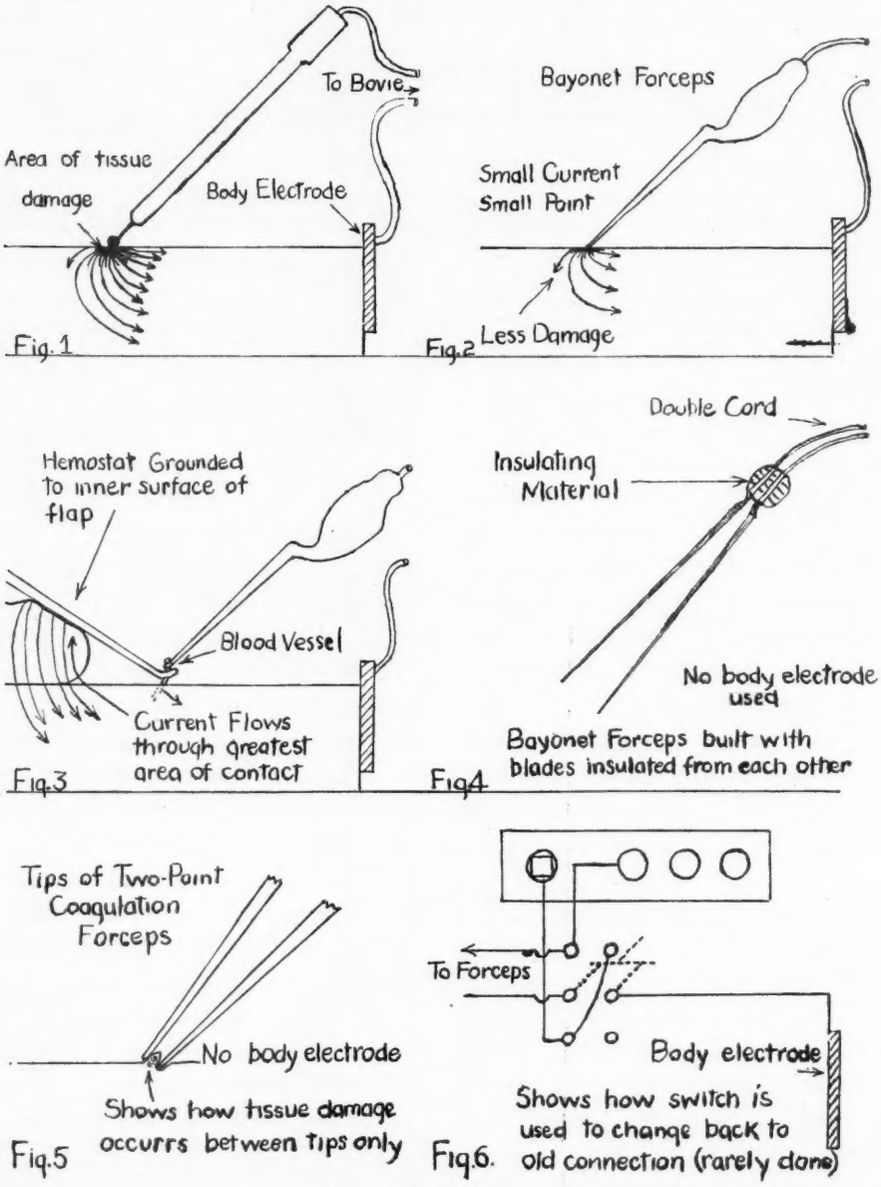
Although there was much improvement we still had a little carbonization at the point of contact unless we were very careful and there was also still some spread of the current a millimeter or so from the point of contact.

A method suggested itself which may find considerable use not only in neurosurgery but in general surgery as well. The body electrode was thrown away and a forceps was constructed (Fig. 4) with the two blades insulated from each other so that the current could pass from one tip to the other, coagulating only the tissues in between. One electrode is connected on the electrocoagulation machine where the patient electrode is usually connected; the

other to the active electrode terminal. While there is a slight spread of current as shown by the diagram (Fig. 5) this becomes minimal when the blades are closed upon a blood vessel.

To use this instrument, vessels must be picked up accurately so that the vessel actually lies between the tips. If the bleeding point cannot be picked up in this manner the tips may be slightly separated ($\frac{1}{16}$ inch) and the coagulation which occurs between will cause hemostasis. Slight carbonization results, not so great as with the one point current even when the greatest precautions are taken.

This method of coagulation has another advantage which we had not foreseen. It may be used where some blood or saline solution surrounds the point of coagulation. This does not seem greatly to hinder its effectiveness, further proof that coagulation occurs only between the forceps blades since salt solution does not produce an appreciable short circuit. As is well known, a fairly dry field (saline sucked away) must be produced with the usual one




Figs. 1 to 6.

point current before coagulation can be very effective.

Another interesting finding was that approximately one-fourth of the usual current was required for most work.

This instrument cannot supplant the usual methods of coagulation since it is rather ineffective, for example, in spraying the inside of a tumor capsule or in spraying a surface which has multiple bleeding points, etc. Here the usual current is superior. Obviously too, the usual body electrode must be used with cutting current. We have used a double pole double throw switch to change from one connection to the other. (Fig. 6.)

The instrument is valuable in cortical dissection and in the dura. In the cortex the vessels may be carefully held between the tips and a small current applied. This causes the faintest white area of dehydration, blood being seen in the vessel to either side. There is no shrinking and no carbonization. The incision may then be made with sharp scalpel through the avascular point. When vessels are met deeper in the cortex they may be coagulated and cut or may be coagulated after cutting by picking up the tips. Here again there is no shrinkage. In the dura there is no noticeable shrinkage and no carbonization. There is also no noticeable effect on the underlying cortex when the dura over it is coagulated.

We have used the two point forceps to stimulate the cortex in attempting to reproduce focal convulsive seizures and in determining cortical localization. They are obviously well adapted to this. The current is, of course, alternating (2 to 5 volts) and the instrument must be temporarily disconnected from the electrocoagulation unit to permit its use in this procedure.

We have used two point coagulation in twenty major neurosurgical procedures and while we do not expect it to revolutionize present methods it is probable that some will find it a useful addition to the present armamentarium.

We were exceedingly grateful to Mr. A. K. Gillis of Pendleton and Arto, Inc. for making us an instrument which is mechanically and electrically excellent and one which is easy and comfortable to handle.

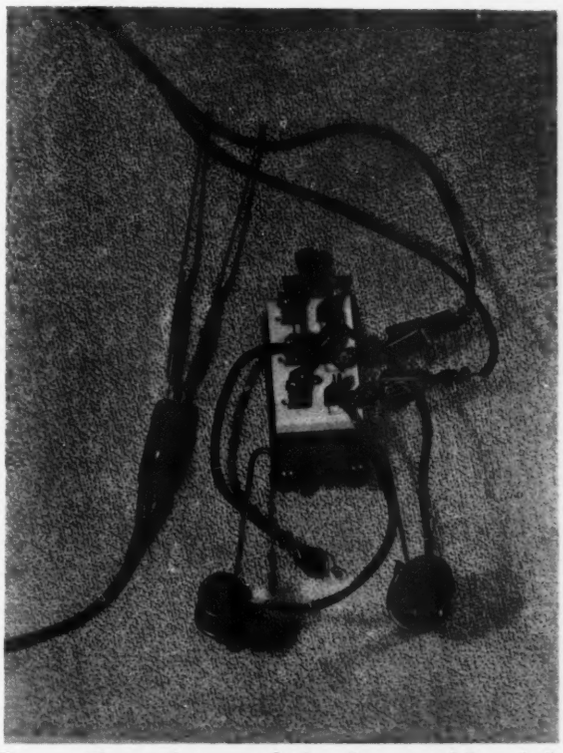


Fig. 7. Two point coagulation forceps with switch which allows transfer from the two point to old one point coagulation.

## CONCLUSIONS

A new and simple principle of electrocoagulation is presented, for use in securing hemostasis in neurosurgical work. An instrument is described which has been used in applying this method.

The essential features of the principle are that the usual body electrode is dispensed with and a forceps is so constructed and used that the current appears only between its tips. Obviously, the closer the tips are placed the smaller the field of electrical activity will be and consequently the smaller the area of tissue coagulated.

The following advantages are claimed:

1. The amount of electrocoagulation current necessary to secure hemostasis is less than one-fourth that required by the usual method.

2. There is less carbonization, tissue dehydration and ensuing tissue reaction.

3. There is no shrinking of the blood vessels being coagulated and current is not carried along a blood vessel into deeper tissues.

4. There is no appreciable shrinking of the dura when a vessel is coagulated on its surface or edge nor is there any noticeable effect on the underlying brain.

5. Coagulation is possible under saline and while washing with saline.

6. Cortical incisions may be made through avascular coagulated points on cortical blood vessels, minimal damage being produced in coagulation since current flows only between the forceps tips. Coagulation occurs only in the vessel held and not in the brain tissue which is adjacent.

A switching arrangement is presented whereby the old method of coagulation may be resumed instantly should the need arise. It is noted that this method should supplement, not supplant the old connection.

We believe this instrument and its connection for "two-point coagulation" should find use in neurologic surgery and possibly in general surgery as well.

AN *artery* is sometimes *pushed forwards* by an underlying growth, and its pulsation in a more than usually superficial position may suggest an aneurism.

# A METHOD FOR REMOVAL OF AREAS OF BRAIN FOLLOWING FREEZING IN SITU*

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THE purpose of this report is to describe a method for removal of areas of brain following freezing in a living animal. The technique consists of delivering the area of brain after freezing with liquid nitrogen. By this method of refrigeration, areas of brain are removed with minimal distortion of tissue and with minimal bleeding. Its feasibility for cortical and subcortical biopsy is apparent, and its use in brain tumor surgery may prove valuable.

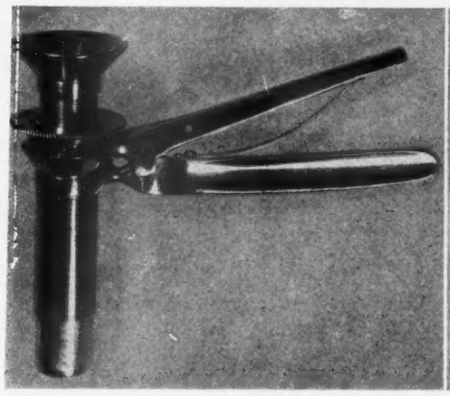

FIG. 1. Illustrates the complete instrument for application to the cortex.

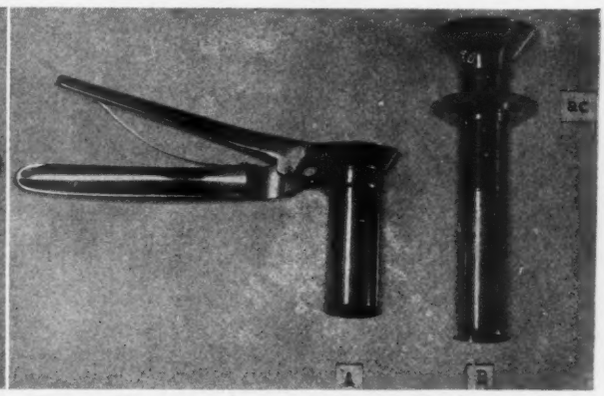


FIG. 2 Illustrates the component parts of the instrument. It consists of two metal cylinders, "A" and "B"; the inner cylinder, "B," is 10 cm. long with a funnel top convenient for pouring, and an adjustable collar (ac). The outer cylinder, "A," 5 cm. long is equipped with a handle and spring lever.

## METHOD

The principle of the method is to apply a freezing agent to the area of brain to be removed, sufficient to freeze it solidly. This permits delivery of the frozen mass in toto without significant hemorrhage. Several available freezing agents were tested; liquid nitrogen was chosen because of its refrigeration properties and its safety. Preliminary observation found the liquid to be sterile.

Cats were used as the experimental animals. The brain was exposed under pentothal anesthesia. In the first group of experiments it was found that pouring liquid nitrogen over an exposed area of brain in the presence of a certain amount of bleeding was ineffective in producing satisfactory freezing, because the liquid nitrogen rolled off the brain without sufficient time elapsing for freezing. An instrument was therefore devised through which the nitrogen is poured and bubbles for a moment over the area outlined for freezing. The tissue is hardened within a few seconds by the rapidly evaporating liquid, and the frozen segment is then removed.

Figures 1 and 2 illustrate the instrument: It consists of two metal cylinders, "A" and "B"; the inner cylinder, "B," is 10 cm. long with a funnel top convenient for pouring, and an adjustable collar (ac). The outer cylinder, "A," 5 cm. long, is equipped with a handle and spring lever. The depth of the segment to be

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removed is determined by the adjustable collar of the inner cylinder. This cylinder is then placed in the outer cylinder and the cylinder is then delivered by pressing the lever arm. The outer cylinder which is adherent to the frozen surface is readily

FIG. 3. Illustrates the instrument in situ in a cat after the freezing agent has been applied.

instrument is ready for application to the surface of the brain. After the cortex is exposed the instrument is inserted to the depth of the previously determined projection of the inner cylinder beyond the outer.

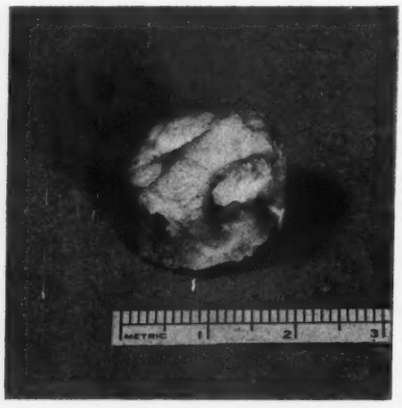


FIG. 4. Illustrates a removed frozen cortical segment.

The liquid nitrogen is then poured into the funnel top causing freezing of the chosen segment within a few seconds. Hemorrhage resulting from the insertion of the instrument is promptly controlled by refrigeration. The instrument in situ is illustrated in a cat. (Fig. 3.) After refrigeration for about one minute the inner cylinder is slowly elevated by turning the adjustable screw which exerts considerable mechanical advantage. The core of frozen tissue captured in the inner lifted from the cortex. The frozen segment removed is illustrated in Figure 4. The slight hemorrhage which usually follows the removal of the instrument is easily controlled by pouring a small amount of liquid nitrogen into the bleeding area. This congeals the blood and a dry crater remains. With the segment removed and hemastasis complete the operative site is ready for closure. Muscle flaps are placed over the crater area and skin sutures complete the closure. The animals exhibited less shock following the removal of the brain tissue by this method than one would expect following the previously used surgical procedures. This problem is under further investigation.

Two of the animals were sacrificed after a month's observation. The crater and surrounding tissue were examined grossly and microscopically. The site of the crater revealed a small amount of old hemorrhage with apparent organization. Microscopic study of the area adjacent to the crater at a distance of about 5 mm. showed small hemorrhages but preservation of the cellular structure. Further pathologic study is in progress concerning the direct effect of freezing on the cortical cells as well as its physiologic effect in such animals. Microscopic examination of the biopsy removed

at operation revealed preservation of cortical architecture.

*Comment.* These animal experiments indicate that refrigeration may be a useful procedure in neurologic surgery. Cortical and subcortical hemastasis can be obtained in a cat by this method. At the depths tested, vessels can be prevented from bleeding before they can be exposed. In its present form the method may be useful for obtaining cortical and subcortical biopsies. Its practical value as an aid in the removal of brain tumors is being tested. It may be possible to simplify the removal of vascular tumors by previously freezing the neoplasm in situ in accordance with the technic described. While this method is by no means intended to supplant accepted procedures such as electrocoagulation, it may prove to be a useful adjunct to brain surgery.

SUMMARY

A method for removal of areas of brain following freezing in a living animal is described. An instrument has been devised for the removal of brain tissue frozen by liquid nitrogen. The use of the procedure in the cat has been demonstrated. The value of the method as an adjunct to neurologic surgery, especially in the removal of vascular tumors, is being tested.

The authors wish to express their thanks to Dr. K. Y. Yardumian of the Montefiore Hospital for his help in the pathological studies; to Ernest Sauerland and Sons of Pittsburgh, for their cooperation in making the instrument; to the U. S. Bureau of Mines, Pittsburgh, for their valuable suggestions and to the Air Reduction Sales Company of Pittsburgh for generously making liquid nitrogen available to us.

# FAILURES IN RHINOPLASTIC SURGERY

## CAUSES AND PREVENTION

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THE school of rhinoplastic surgery founded by Professor Jacques Joseph disappeared in great measure with the advent of the Third Reich, when he became an exile and died shortly thereafter. Teaching facilities in this branch of surgery became restricted because only a few men were able to associate themselves with the small group of Joseph's pupils in this country.

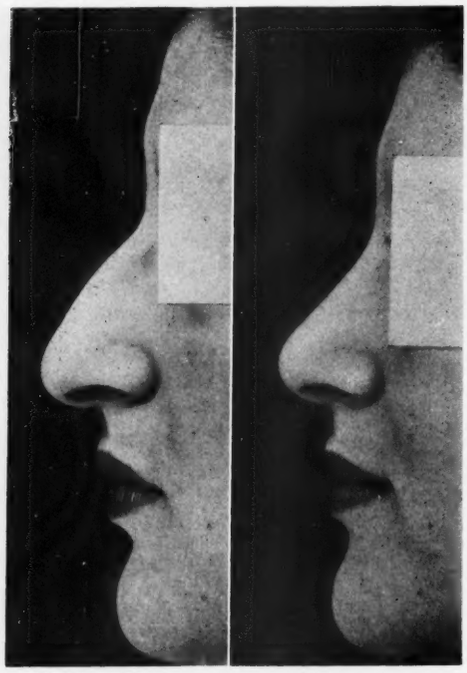

FIG. 1. FIG. 2.
FIG. 1. Convex profile.
FIG. 2. Following correction.

Lectures, scientific publications and demonstrations of patients during a decade or more have convinced the medical profession that a profound psychologic factor is involved when a nasal disfigurement is either properly or improperly corrected. When rhinoplastic procedures gained the approval of the medical profession, the demand for the correction of nasal and other facial disfigurements became increasingly great.

Plastic surgery, and rhinoplasty in particular, present a dual problem. First, the technique must be mastered as in all surgical procedures; second, equally or even more important, is the art of creating "form" which has a definite esthetic value. Success in rhinoplastic surgery, therefore, depends upon a combination of surgical training and an innate creative ability. Because the essential artistic instinct is a personal faculty, it accounts for the varied attainments of even well trained men.

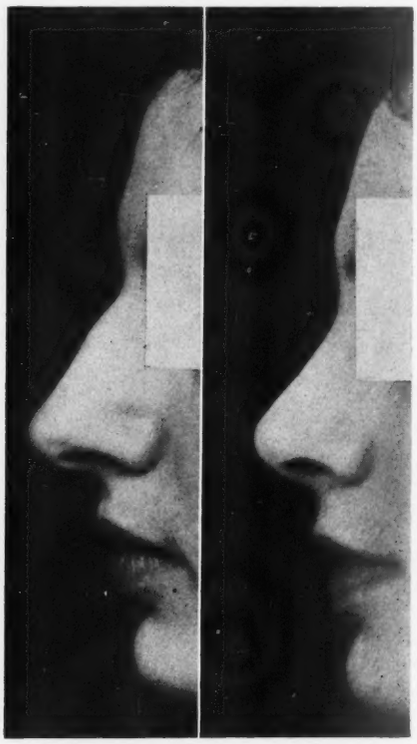

FIG. 3. FIG. 4.
FIG. 3. Prominent profile.
FIG. 4. Following reduction.

The admittedly large proportion of unfavorable results following rhinoplastic operations is a definite indication that the

majority were done by men lacking in one or the other of the essential qualities. Some results show ample evidence that both prerequisites were lacking. The patients often suffer secondary surgical disfigurements, varying in degree from an unnatural appearance to actual mutilations. It is difficult to exaggerate the great injury inflicted and the social and economic loss.

The patient who submits himself to a rhinoplastic operation does so with the expectation that the disfigurement which is a hindrance to economic or social progress will be eliminated and a normal nose, conforming to general esthetic conceptions, will result.

The esthetically normal nose is in proper proportion to the other facial features in length, breadth, and profile elevation. An important element of a satisfactorily reconstructed nose is that it must not show evidence of having been operated upon. One must particularly avoid what is commonly known as a "surgical nose."

The first group of cases illustrated here shows the various types of nasal disfigurements before and after correction. Particular attention is called to the normal appearance of the corrected noses and especially to the fact that the nasal tip retains the normal esthetic curves provided by nature. (Figs. 1 to 8.)

An analysis of rhinoplastic failures shows that they usually fall into one or more of three groups. The secondary surgical disfigurement involves either the upper, middle or lower third of the nose. The individual steps of a complete rhinoplastic operation can be considered and the more common errors which lead to unsatisfactory, and sometimes truly amazing results, can be pointed out. This study is based on my experience with a large number of patients who sought physical and emotional relief from an unsatisfactory rhinoplastic operation or from a secondary surgical deformity.

1. *The Upper or Bony Third of the Nose.* In this category the surgeon is usually concerned with the removal of a nasal hump, or the lowering of the profile line and the narrowing of the nasal bridge.

The errors commonly committed which

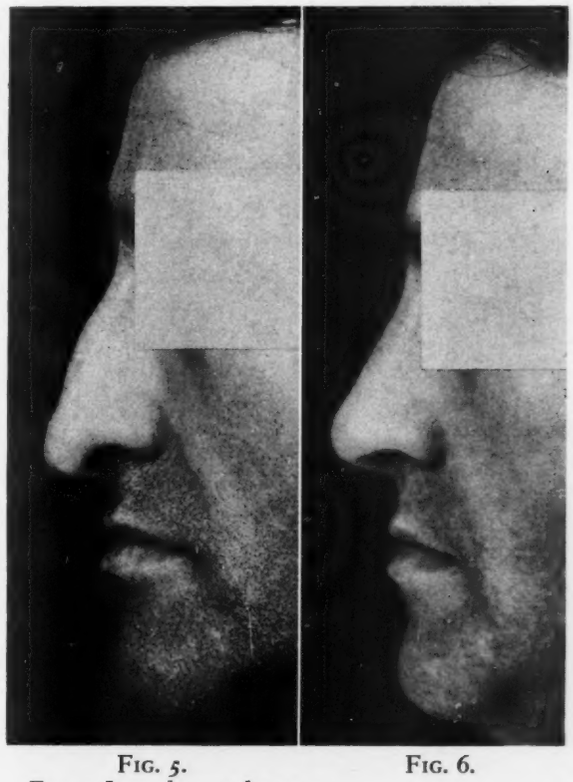

Fig. 5. Fig. 6.

Fig. 5. Long humped nose.
Fig. 6. Normal proportion following correction.

lead to poor results are as follows: (*a*) the removal of bony structure without a compensative reconstruction of the rest of the nose, resulting in a bird-beak effect (Fig. 9); (*b*) cutting through the right and left side of the nasal hump at unequal levels, producing a flattening of one side or the other; (*c*) failure to cut the dorsal edge of the septum down to the same level as the lateral walls, producing a "razor edge" nasal dorsum instead of one with a normal width; (*d*) failure to narrow the nasal bridge, resulting in a flat dorsal plateau with retraction of the skin into the hiatus created by the removal of the hump (Fig. 10); (*e*) failure to produce a complete fracture of the lateral walls, tending to widen the nasal bridge; (*f*) deviation of the bony septum, preventing a proper "in-fracture" of the affected side, resulting in a scoliosis; (a deviation of the bony septum must al-

ways be corrected prior to any attempted narrowing of the nasal bridge) (Fig. 11); (*g*) cutting through the frontal processes of the superior maxillae at an improper level, producing a step-like thickening at the base of the nose.

Fig. 7. Long nose with wide hanging tip.

Fig. 8. Normal appearance of tip following correction.

Fig. 9. Bird beak effect due to incomplete operation.

The surgeon who undertakes to perform rhinoplastic operations must realize from the foregoing axiomatic statements that an exacting technique is of prime importance and that any slight deviation therefrom or error of judgment regarding measurements, proportions, or position, will produce a disappointing result.

2. *The Middle Third of the Nose.* This consists of the lateral cartilages and the septum.

A nasal hump, or an excessive profile elevation, is rarely confined to the bony structure alone. The cartilages of the middle third of the nose are usually an integral part of the nasal hump and these structures must therefore be brought down to the newly created level of the bony part.

Errors are: (*a*) failure to level the septum to the required profile line, leaving a cartilaginous hump or a dorsal convexity (Figs. 12 and 13); (*b*) the lateral cartilages must be trimmed down to the newly leveled septum, because failure to carry out this step leaves an elevation on one or the other side of the midsection of the nose; (*c*) the lower portion of the lateral cartilages must be trimmed in order to reestablish a normal relationship between the lateral and alar cartilages. If this trimming is not carried out an excessive thickening of the nasal tip results, with an obliteration of the nasal vestibule. (Fig. 14.) It is important to bear these points in mind in order to avoid an unsightly secondary disfigurement after an otherwise properly performed nasal correction.

The necessity for a secondary correction following a primary rhinoplasty is not always an indication that the original correction was improperly performed, provided no surgical disfigurement results.

The conservative surgeon makes a sufficient allowance for the normal dropping of the nasal tip after the primary shortening. But the exact degree of dropping cannot always be anticipated, because the muscular action and tonus of the labial muscles vary in different individuals. (Fig. 15.)

A secondary shortening of the nose is indicated in about 10 per cent of patients.

Other secondary corrections which may aid in the final satisfactory result are, either an additional narrowing of the nasal tip or of the nostrils.

Fig. 10. Failure to narrow nasal bridge.

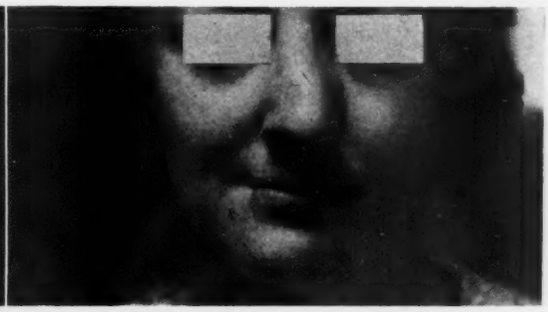

Fig. 11. Failure to correct septal deformity prior to plastic correction.

3. *The Lower Third of the Nose.* It will be obvious from the foregoing that great caution is necessary in corrections involving the lower third of the nose. It is in this area that the largest number of surgical disfigurements occur. The cartilages comprising the lower third of the nose have, aside from other functions, one definitely esthetic. The cartilages may be too large or disproportionate. Faulty technique or disregard of the anatomic functions of the cartilages will always result in a distinct surgical disfigurement. We see more patients with surgical disfigurements or mutilations of the nasal tip, than with any other type of secondary rhinoplastic deformity.

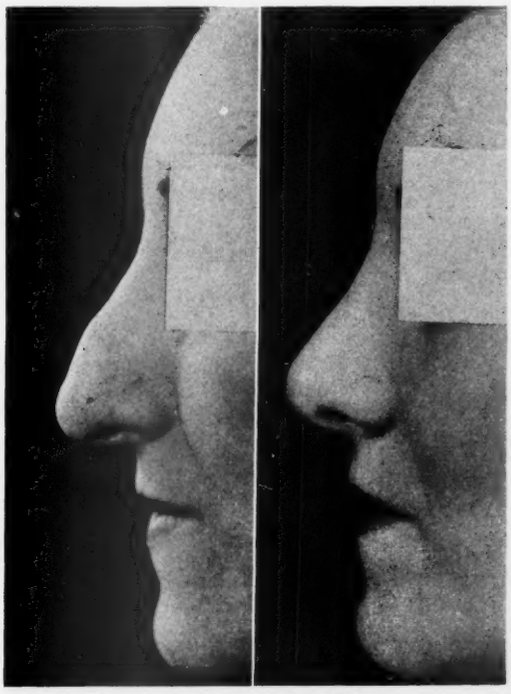

Fig. 12. Fig. 13.

Fig. 12. Failure to level cartilaginous hump to level of bony correction and insufficient shortening of nose.

Fig. 13. Same patient following secondary operation.

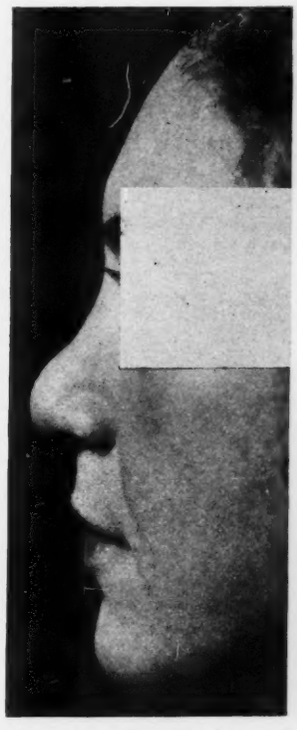

Fig. 14. Lower end of lateral cartilages curled up and producing a thick nasal tip.

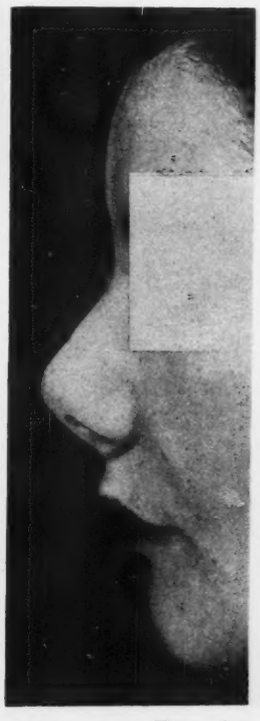

Fig. 15. Excessive shortening of the nose.

The end result in the correction of these mutilations depends upon the degree of destruction of vital nasal tissues. When the disfigurement is slight a considerable

measure of improvement can be brought about, while the more flagrantly distorted noses are almost beyond repair.

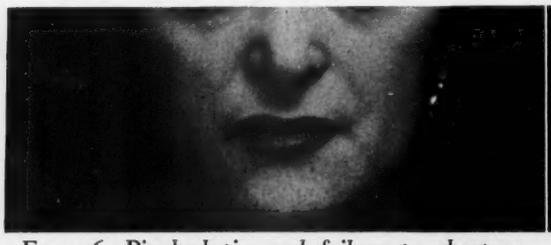

FIG. 16. Pinched tip and failure to shorten hanging columella.

FIG. 17. Shrinkage of tip due to complete removal of alar cartilages.

I have described[1] the most satisfactory techniques employed in corrections involving the lower third of the nose to avoid the most common errors.

(*a*) In shortening the nose, the watchword should be *conservatism*. The nose should be shortened to an extent somewhat

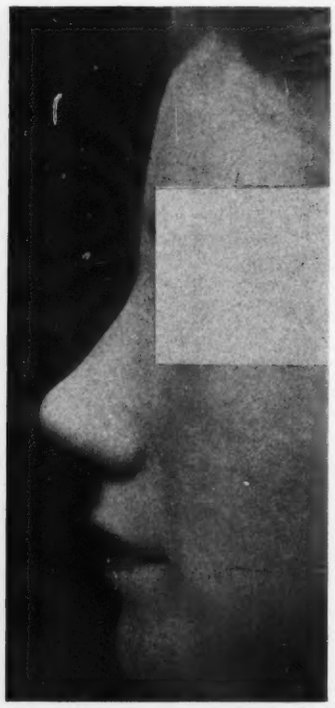

FIG. 18. Retracted columella.

less than the length of the chin line. The usual amount of postoperative droop is about 2 mm. When it exceeds this, it is preferable to carry out a secondary shortening, following which there is usually no postoperative dropping of the nasal tip.

(*b*) The narrowing of the nasal tip should be carried out with the objective in mind that sufficient alar cartilage and mucosa must remain to afford proper support for the overlying skin. The commonly observed pinched-tip effect will thus be avoided. The alar cartilages should never be removed completely. (Figs. 16 and 17.)

(*c*) A rather common surgical disfigurement is a retracted columella. This effect is produced by the removal of the posterior surface of the columella while shortening

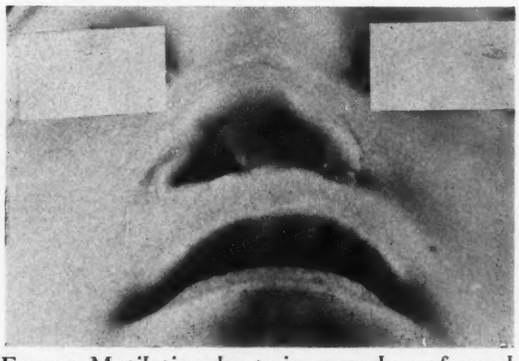

FIG. 19. Mutilation due to improperly performed Weir operation.

the nose. (Fig. 18.) Such a deformity is not easily remedied because a supporting structure has been lost. It seems to me that the error is made with the assumption that a "hanging septum" can be corrected by removing the posterior half, or more, of the columella. As a matter of fact the "hanging septum" is merely an excessive convexity of the anterior edges of the columellar cartilages. When this condition is present it calls for specific correction, the technique for which is described in my book,[1] as well as in Professor Joseph's "Die Rhinoplastie."[2]

One step in a general rhinoplastic correction which requires careful consideration is the reduction of the size of the nostrils

by the excision of a triangular section, anteriorly or posteriorly. During the operation one may get the impression that the nostrils appear larger than before. This is frequently due to the novocaine infiltration, and subsides after the solution is completely absorbed. I have observed many instances where the base of the nose, in a previous operation, was unnecessarily narrowed, resulting in a typical adenoidal facies. Unless one is certain that this step is indicated it is best to defer it until all swelling has subsided. It will then rarely be found necessary to reduce the size of the nostrils. As a rule, the narrowing of the nasal tip, according to the technique

Fig. 20. Mutilation of nasal tip due to improper surgical technique.

Fig. 21. Same patient following a series of corrective operations.

Fig. 22.

Fig. 23.

Fig. 22. Profile view of same patient in Figures 20 and 21.
Fig. 23. Profile view following correction.

Fig. 24. Mutilation of nasal tip due to improper surgical technique. Front view.

Fig. 25. Same patient following a series of corrective operations.

described in my book, will diminish the size of the nostrils sufficiently to make the second operation unnecessary.

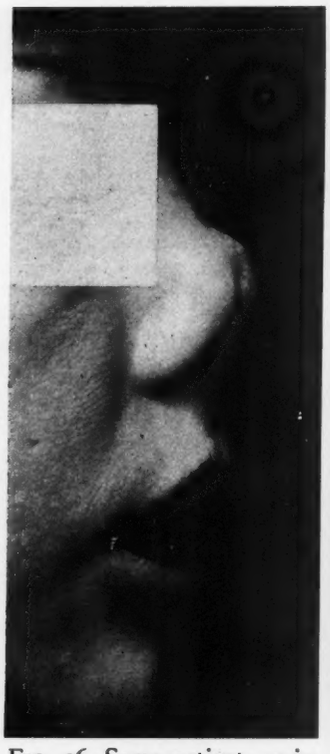

FIG. 26. Same patient, as in Figure 24, profile view prior to secondary correction.

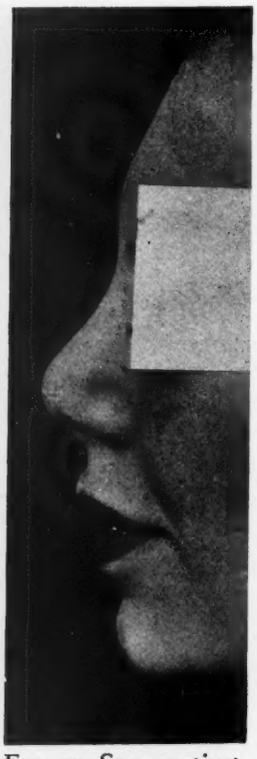

FIG. 27. Same patient profile view following secondary correction.

Another alar operation which is far too frequently performed is what is commonly called the Weir operation. It consists in the excision of a crescentic section of the alae at their attachments. This is indicated only in persons with true negroid nostrils. In all other instances which I have observed, this operation flattens the nostrils and obliterates the nasolabial fold. It constitutes a surgical deformity which, as far as I am aware, has not been successfully corrected by a secondary operation. (Fig. 19.)

## CONCLUSIONS

I ardently advocate conservative surgery even if the necessity for a slight secondary operation should arise and if the esthetic requirements of the patient demand it. It is obvious, however, that the primary operation must produce a decided improvement in the patient's facial appearance.

When the primary operation is unskillfully performed and a definite surgical disfigurement is produced, it may require a series of secondary operations, each more difficult and complicated than the original one before the patient can be restored to economic usefulness, or saved from a state of physical depression and a life of social ostracism. Unfortunately, some of these patients are so severely mutilated that they cannot return to their gainful occupations, especially if a "presentable appearance" is requisite. Figures 20 to 29 are examples of severe surgical mutilations of the nasal tip and the results obtained after a series of corrective plastic operations.

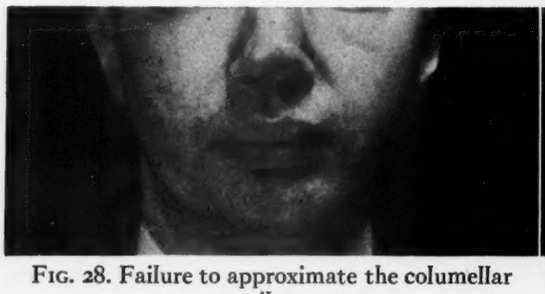

FIG. 28. Failure to approximate the columellar cartilages.

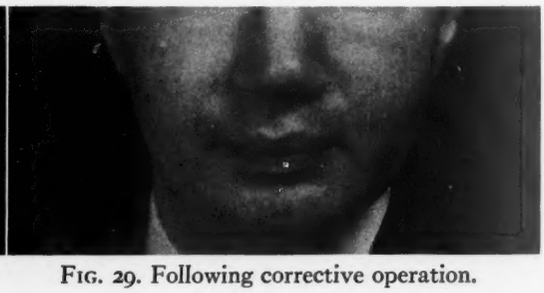

FIG. 29. Following corrective operation.

## REFERENCE

1. SAFIAN, J. Corrective Rhinoplastic Surgery. New York, 1935. The American Journal of Surgery, Inc.
2. JOSEPH, JAQUES. Die Rhinoplastie.

# THE SURGEON AND COLONIC ALLERGY*

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EVERY surgeon of long experience can recall patients who seemed to require immediate operative interference because of typical symptoms of an acute abdomen and who, when the abdomen was opened, showed only an edema of the intestinal walls which seemed to subside during his manipulations, or an erythema for which no definite surgical treatment seemed to be indicated. Also, unfortunately, many patients have been explored to determine the reason for indefinite but troublesome symptoms, nothing very definite was found and the abdomen closed, or a routine appendectomy performed. Many of these mistakes are made because in the preoperative study the possibility that the symptoms might be caused by an allergic reaction has been overlooked. It is important for the surgeon as well as the internist to become "allergy-conscious" so that embarrassing or even dangerous situations of the type just described may be avoided. Laymen have long recognized the fact that certain otherwise nourishing foods would invariably produce bowel symptoms and have avoided them, or, as is so frequently the case in sensitization to milk, have used the particular food for cathartic purposes. In recent years much has been written about allergy, a special nomenclature for describing its phenomena has been developed, and much experimental work has been carefully carried out, and yet it is a fact that many perfectly obvious cases are constantly being overlooked by the general practitioner and the specialist.

The cause of allergy has not been determined. Its hereditary transmission along Mendelian lines has been amply proved. We know that in some way the endocrine glands play a part in the tendency to allergy, experiments with extracts of adrenals, the pituitary and the parathyroids having demonstrated that these glands and perhaps others influence allergic manifestations. Autonomic imbalance has been described as an etiologic factor. We have seen, in some cases, a complete disappearance of allergic symptoms after patients have had a thorough eradication of all focal infections, have moved from one climate to another or have suffered from a febrile disease. The seasonal occurrence of symptoms, not only because of exposure to or ingestion of substances peculiar to certain seasons, but often apparently because of a change in body metabolism, has been observed, and in some cases deficiency states, especially subvitaminosis, have seemed to play an important part in the condition. Trauma has also been considered an important factor in initiating sensitivity. Just as the etiology of allergy is apparently so varied, even the nature of the actual substances producing the symptoms is still a subject for disagreement. It is now generally considered that a specific protein is the cause, although some have in the past insisted that split proteins produced the phenomena. Recently histamine has been implicated. Drugs and chemicals have been known to produce manifestations exactly like those caused by protein sensitivity and even "physical allergy," with reactions to light, heat, cold and electricity has been demonstrated. The problem of bacterial allergy is being studied and seems to involve some changes in our conception of infection and immunity. The fact that areas of skin or mucous membrane may be sensitized to specific proteins by intracutaneous or intramucosal injections of serum of persons

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sensitized to those proteins is also an interesting observation.

The reactions caused by allergy in the colon are directly observed by the surgeon at proctoscopy or operation and indirectly by the roentgenologist in the course of a gastrointestinal study. In 1925,[1] I suggested that the reactions could be shown to parallel the then better known changes in the respiratory tract, with mucosal and neuromuscular manifestations. The mucosal reactions then described were hyperemia, hypersecretion, edema, often followed by local ischemia, necrosis, sloughing and ulceration, hemorrhage or perforation, and if chronic or repeated, by induration, fibrosis and adhesions. Submucosal hemorrhages, producing the petechial or purpuric areas often seen on proctoscopy, may break down and bleed. The neuromuscular reactions were described as consisting of motility disturbances, spasms and dilatations. In recent years Walzer, Gray and Harten and their co-workers[2,3] have demonstrated allergic reactions experimentally in the human rectum, and in the ileum and colon at the site of colostomies. In Rhesus monkeys they have produced reactions in the intestines, have removed specimens during the reactions and have demonstrated the microscopic findings. They passively sensitized areas of mucosa of rectum, ileum or colon simultaneously with an area of skin on the arm, by injecting into these sites small amounts of serum from a person sensitive to peanut protein. Then on feeding a peanut mixture by mouth, instilling it into the bowel or applying it locally, they observed the reactions. In the skin these consisted of the usual urticarial wheals. In the mucosa they usually consisted of excessive secretion and pallor, followed rapidly by a steadily increasing edema and marked hyperemia, the reaction still being present after two hours and then gradually subsiding. These local reactions began to appear in from five to eight minutes after the administration of the antigen, proving its rapid absorption in the unchanged state, even when given by mouth. Obviously it was not advisable to repeat the experiments at sufficiently close intervals to produce more severe or chronic changes, but these can be readily imagined. Microscopic changes observed in specimens of stomach and bowel of monkeys removed during an allergic reaction consisted of edema, more marked in the deeper layers, especially the muscularis mucosae, and of round-cell infiltration (usually eosinophiles) and beginning interstitial changes. In 1937 Kaijser[5] resected a piece of human small intestine during an acute allergic reaction and demonstrated marked edema and eosinophilic infiltration. Appendices removed in acute appendicitis are often found to be infiltrated with eosinophiles.

*Mechanism.* Experimental work confirms what clinical observation has long led us to believe, that the reactions in the gastrointestinal tract are caused less frequently by direct contact of the food with the mucosa than by the irritation from absorbed unchanged protein reaching the organs through the blood stream. While absorption of unchanged protein from the upper part of the gastrointestinal tract has been shown to be very rapid and to occur independently of the amount and character of the gastric secretion, it has long been felt that gastric anacidity may enhance the speed of such absorption. The addition of solvents to the food, notably alcohol, may also cause more rapid absorption and in some patients has been known to be necessary to the production of a reaction.

*Symptoms.* Allergic reactions in the colon such as have just been described would, according to their severity, produce a variety of symptoms. The milder early reactions, with erythema and hypersecretion as a prominent factor and motility disturbance or spasm as a complication, would produce the symptom complex usually described as "mucous colitis" or the equally indefinite syndrome now called "irritable colon." A more severe reaction could be called colon spasm or colitis, or

with more marked edema and thickening could produce a picture of acute intestinal obstruction or an acute inflammation or perforation. The most severe mucosal reactions, starting with edema and erythema followed by marked hyperemia and infiltration, by herpetiform rashes in the mucosa, and subsequent sloughing and denudation of the mucosa, present the picture of so-called "idiopathic" ulcerative colitis. In the anal region, any of these changes, even those showing only mild hypersecretion and edema, are usually associated with more or less marked pruritus, and in my opinion most cases of this distressing condition can be traced to an allergic cause. Some of the very marked anal and rectal conditions in which infiltration, ulceration and bleeding are found, have been shown to be allergic in origin. Intermittent gross hemorrhages, due to purpuric areas in the rectum or sigmoid, may frequently be traced to a food sensitization, and in these cases the presence of skin purpura during the attacks may be a clue to their origin.

*Diagnosis.* The diagnosis of allergy as a cause of colonic manifestations requires a careful study. It is only after a careful and complete gastrointestinal check-up has ruled out actual organic lesions or bacterial or parasitic invasions that we should be prepared to consider allergy as the sole or primary cause of the symptoms. Whereas in the past many patients have been incorrectly treated or uselessly operated upon because allergy was not recognized, there is grave danger today that the clinician who jumps at a diagnosis of allergy may be mistreating a patient with a definite infection such as a specific colitis or may be dangerously postponing operation for a malignancy. Even though the diagnosis of allergy may be correct, a more serious lesion may be masked by the more spectacular allergic symptoms, so that the importance of a complete gastrointestinal survey, with detailed history, gastric analyses, stool examinations and gastrointestinal and colonic Roentgen studies cannot be overemphasized. The principal points in making the diagnosis follow:

1. The *history* constitutes the most important single diagnostic aid. Allergy may be suspected when any of the following facts in regard to the patient have been ascertained:

(*a*) A family history of allergy, not necessarily of the same type as that found in the patient, but a story of asthma, hay fever, migraine, eczema, urticaria or any other periodically recurring illnesses in ancestors, brothers and sisters or even children. Allergy does not commonly produce the same manifestations in the different members of a family, although the Mendelian law seems to be followed so far as occurrence of allergy in general is concerned.

(*b*) A previous history of definite allergic symptoms in the patient's earlier life. The previous occurrence of "bilious attacks" (migraine) and periodic diarrheas, respiratory or skin manifestations, either previously unexplainable or definitely attributed to certain foods or inhalants may be taken as evidence of an allergic tendency.

(*c*) The onset or cessation of symptoms at puberty or the menopause, during pregnancy or after some great emotional stress is also very suggestive.

(*d*) The observation by the patient that the ingestion of certain foods is always followed by certain definite symptoms. Among the more common gastrointestinal symptoms thus described are the diarrheas following the drinking of milk ("milk acts as a cathartic"), heartburn or "repeating" after certain foods and the abdominal cramps usually occurring after eating certain mixtures like fruit salads, salad dressings or soups, one or more ingredients of which may be the causative factor.

(*e*) The occurrence of symptoms on a certain day of the week in persons living in institutions or in persons whose eating habits are so systematic that certain foods are always taken on certain days of the week. "Blue Monday" is often caused by

the fact that only on Sunday does the family eat chicken, and on Monday the members of the family who are sensitive to chicken suffer from migraine or diarrhea.

2. *Physical examination* may be of little or no assistance in making the diagnosis. Where other than colonic manifestations are present, the nasal mucosa may show the changes found in hay fever or vasomotor rhinitis, the eye may show hyperemia and even corneal herpes, the chest may be emphysematous from asthmatic attacks, the skin may show one or more of the many lesions known to be allergic in origin. Abdominal examination may disclose nothing of importance, may indicate some spasm of the colon or in the severe cases may show all the signs of an acute abdomen, simulating either an acute inflammatory or perforative lesion or an intestinal obstruction. In some cases the very rapid and complete disappearance of the symptoms and signs in these acute cases after the administration of a dose of adrenalin or ephedrine intramuscularly or sublingually often will give dramatic evidence of the allergic nature of the attack.

3. *Laboratory examinations* are of indirect value. Gastric anacidity may or may not be found. Excessive secretion of gastric mucus may be an indication of continued gastric irritation. Pylorospasm may cause temporary delay in gastric emptying. Stools may show excessive mucus in the milder cases, blood, mucus and even pus in the severe cases. Eosinophiles in the rectal discharges and marked eosinophilia shown in the blood count are very suggestive findings, especially when evidence of protozoal infestation can definitely be ruled out.

4. *Roentgen examination* may be of considerable value in the diagnosis of colonic allergy. In the gastrointestinal series, if the barium meal contains some substance like chocolate or acacia, to which the patient's gastrointestinal mucosa is sensitive, there will be noted mucosal puckerings or rugal coarsening in stomach and duodenum, and spasms and mass contractions, rapid "writhing" peristalsis or actual reverse peristalsis in the duodenum. With the hypermotility the meal may reach the colon more rapidly than normally and evidences of colonic hypertonicity and hypermotility may be observed. In the opaque enema study, spasms and hypertonicity may be noticed and at times, where the enema contains offending substances, the mucosal changes may be seen by fluoroscopy or films. The well-known findings in ulcerative colitis, consisting of "fuzzy" outline, hypertonicity, obliteration of haustra and later deformities and defects, are quite marked and may be shown gradually to disappear as the condition improves.

5. The *determination of the allergic factor causing the reactions* is the most important part of the diagnosis. While at times history of occurrence of symptoms at the menstrual epoch will indicate a sensitivity to some hormonal factor produced at that period, their occurrence coincident with activity at some infective focus may suggest bacterial allergy or their occurrence after medication may indicate chemical or drug idiosyncrasy, most cases of colonic allergy are probably due to sensitivity to specific food proteins. At times the recognition of the cause of the symptoms is comparatively easy, as in the case where the patient has previously known of the cathartic effect of milk and has continued to take daily small quantities, sufficient, however, to produce continuous bowel irritation. One self-evident principle in regard to the determination of the food is that where the symptoms are continuous and chronic, the food must be one taken daily or very frequently, whereas intermittent attacks must be due to foods taken on more rare occasions. Attacks lasting a single day must be due to a single ingestion of the food, attacks of two or three days' duration to the eating of left-over or made-over dishes containing the food for a couple of days. Skin sensitization tests are of some value in determining the foods responsible for the reaction,

but it has been shown that foods giving skin reactions by the scratch, intradermic or patch methods, may not necessarily be concerned with any other than skin manifestations, and not always with these. In the determination of food allergies they have been practically abandoned. In the end the only real test is the keeping of careful records of repeated trials of a suspected food in order to be convinced of its effect. Various "elimination" and "addition" diets to aid in the determination of the offending foods have been suggested, the former consisting of fairly full diets in which daily changes or eliminations are made until symptoms clear up, the latter starting with nothing but water, or possibly the old Duncan Bulkeley rice and water diet, producing a cessation or marked alleviation of symptoms, and adding foods one or two at a time until reactions occur. The latter is obviously not suitable in cases where the patient is much in need of immediate nourishment. In any test diet it is necessary to get a complete cessation or marked alleviation of the symptoms in order that the detailed record of the diet taken during and preceding the period of relief may be compared with the foods taken during exacerbations. In general it may be said that a food to which a person is sensitive will always produce symptoms, even when taken in small quantities, so that if no symptoms occur after even a single ingestion of a food, that particular food may be absolved of blame for previous or subsequent symptoms. A possible exception to this rule might occur where sensitization to two or more single foods might be slight and the reaction hardly noticeable, but when these foods were taken at the same time a more severe reaction might occur.

In an effort to simplify the dietetic study, we have been using, in the past couple of years, two test diets, which have been of considerable help. Test Diet Number 1, containing foods usually taken daily by the average individual, is of value as a starting diet in cases in which symptoms do not occur daily or continuously, and consists of only five foods: milk, egg, wheat, potato and orange (or where orange is known to be an offending factor, some other fruit may be substituted for it). These foods can be used to make a fairly satisfactory though monotonous diet as follows:

TEST DIET NO. 1

*5 Foods:* Milk, Egg, Wheat, Potato, Orange

*Breakfast:* Milk, egg, wheat cereal and cream, wheat bread and butter, and orange.

*Lunch:* Milk, egg or cheese, potato, bread and butter, dessert (orange-flavored custard or wheat pudding or whole orange).

*Supper:* Same as lunch or breakfast.

*Between meals and at bedtime:* milk with cream and glucose and wheat crackers.

On such a diet, three possible things may occur: (1) the patient may show no improvement, indicating either that the offending food is contained in the diet or that diet has nothing to do with the symptoms; (2) the patient's symptoms may be much exaggerated, indicating that one or more of the ingredients of the test diet are causing the symptoms; (3) there may be an alleviation of the symptoms and signs, or sometimes, in milder cases their dramatic cessation, indicating that the diet contains none of the foods causing the symptoms. If the third result is obtained, after a few days of this diet new foods are added one or two at a time, a record kept of the results and suspicious foods rechecked. If symptoms are the same or increased, one of two procedures is indicated, namely, the elimination of one of the five foods at a time until improvement is noted, later rechecking the foods suspected, or, perhaps what is even more advisable, the substitution of a test diet containing five foods, none of which have been present in the first diet. This diet we call *Test Diet Number 2*, and it consists of rice, rye, gelatin, one vegetable and one fruit. A sample diet of this kind would be as follows:

TEST DIET NO. 2

*5 Foods:* Rice, Rye, Gelatin, 1 Vegetable, 1 Fruit

*Breakfast:* boiled rice, rye krisp, gelatin drink, raspberries.

*Lunch:* Peas, rice, rye krisp, gelatin drink, raspberries.
*Supper:* Same as lunch or breakfast.
*Between meals and at bedtime:* Gelatin drink and rye krisp.

*Note:* The gelatin drink is made by adding a heaping teaspoonful of pure powdered gelatin to a fruit juice made of the fruit or berry used in the diet, sweetened and fortified with glucose. Butter does not contain sufficient milk protein to cause any reaction as a rule and is a valuable addition to this restricted diet. The procedure to be followed is the same as that described for the first test diet, and as a rule the offending food or foods can rapidly be ascertained.

*Treatment.* Aside from appropriate endocrine therapy, the eradication of all infective foci, local treatments which may seem indicated, or the use of sedatives and psychological treatments in patients who require it, the essential factor in the care of an allergic patient is to eliminate all offending foods from the diet. If the foods to be eliminated are those not ordinarily necessary to the enjoyment of a natural, well-balanced diet, this elimination is easily accomplished, and the patient can continue for the rest of his life, if necessary, without taking any of these foods. If, however, they are articles which occur in the normal daily diet, such as milk, egg and wheat, it is much more difficult to avoid foods containing them, and desensitization to these foods may be attempted. To do this it is not necessary to start with hypodermatic injections of the pure protein extracts of the food. Oral desensitization is entirely satisfactory. It is best to start with minute quantities each day, increasing the quantities gradually until symptoms reappear and finally arriving at a dosage beyond which the patient cannot go without getting symptoms, but usually large enough to permit of the taking of food mixtures containing small amounts of the food, as in the case of milk in cereal or coffee, egg in pudding or ice cream, or wheat in crackers or cake. In the case of milk, the initial dose in beginning desensitization should be one drop of whole milk, in the case of egg, one drop of a solution of a beaten egg in 8 ounces or more of water, in the case of wheat, a small pellet made of bread. In this connection it is well to remember that when desensitization has been attained, the patient must take the acquired dosage of the food every day, as omission of the food for even a few days will reëstablish the sensitivity. This fact makes it undesirable to desensitize any patient to a food which he would not care to eat each day or could not obtain at different seasons. Nonspecific desensitization by the use of peptone intravenously or by the employment of various forms of artificial pyrexia has been fairly successful in some cases, but in recent years has been rarely employed. The injection of increasing doses of histamine has also at times accomplished a nonspecific desensitization.

In the *treatment of an exacerbation of acute symptoms*, such as an attack resembling an acute abdomen, or an acute intestinal obstruction, or in a very severe case of ulcerative colitis, it may be necessary to institute measures not ordinarily required in the more chronic case.

1. Frequently a hypodermatic injection of 1 c.c. or less of adrenalin chloride solution, 1:1000, will give immediate and almost miraculous relief of symptoms, although at times it may cause such distressing symptoms of thyroid stimulation as to make its use undesirable. At times pituitrin will give the same relief, but it may unduly stimulate peristalsis and thus, if a real acute abdomen were present, would do harm. Parathyroid hormone with calcium may also be of value. Histaminase has recently been used with dramatic effect in some cases, but is not constantly reliable.

2. Removal of the offending factors as rapidly as possible would seem to be most desirable. In milder cases, a dose of castor oil or even a lavage or a cleansing enema may be used as a first step. However, as absorption of the allergen is probably the

most important cause of the reactions, it is usually noted that reactions cease when further absorption does not occur because all present in the meal has been absorbed and because unabsorbed portions have become dehydrated in the lower bowel and therefore are no longer capable of absorption. I have seen patients develop secondary reactions when a saline cathartic was taken after subsidence of an attack, probably because of the liquefaction and absorption of protein which had been previously dry and unabsorbable. This is probably why castor oil, with its detoxifying as well as cathartic effect, is most desirable in these cases.

3. In order to keep up nutrition, restore normal gastrointestinal function, and avoid general dehydration, it is important to feed the patient early and frequently. A well-balanced diet should be administered as soon as the offending foods are known, careful choice of substitutes for these foods being imperative. Attention to adequate vitamin and mineral content is necessary.

*Prognosis.* The prognosis in any case of allergy is not good for cure. We know that sensitivity to a given food may never disappear or may suddenly cease spontaneously, only to be followed by sensitivity to another, previously innocuous food. We know that during and after pregnancy or following fevers there may be temporary and even at times, prolonged relief from symptoms, and on the other hand that puberty, pregnancy or the menopause may be periods when allergic manifestations may first and only be observed. It is also well to bear in mind that chronic irritation, due to the constant eating of foods to which a patient is sensitized may possibly be a factor in the etiology of cancer, and further study along this line may indicate the importance of the recognition and treatment of allergy in the prevention of cancer. Already it is realized that the polyposis and fibrosis following ulcerative colitis form a soil in which carcinoma may develop.

## SUMMARY

1. It is important that surgeons as well as general practitioners recognize the common allergic reactions in the colon.

2. Mild allergic reactions cause mucous colitis, mild spasms and "irritable colon," more severe mucosal reactions are the cause of ulcerative colitis.

3. Very severe edema and spasm may produce symptoms of acute intestinal obstruction or an acute inflammation, hemorrhage or perforation.

4. Allergy should not be made the primary diagnosis in any case until all organic causes for the symptoms have been ruled out by complete gastrointestinal and general study.

5. The primary treatment of allergy consists essentially of the elimination of the offending factors and of general hygienic care.

6. Desensitization, specific or nonspecific, and the treatment of endocrine, neurologic and metabolic disturbances, and removal of focal infections are necessary.

7. Allergy as a precursor of cancer deserves further study.

## REFERENCES

1. Andresen, A. F. R. *M. J. & Rec.*, Sept. 2, 1925.
2. Gray, I. and Walzer, M. *Am. J. Digest. Dis. & Nutrition*, 4: 707, 1938.
3. Walzer, M., Gray, I. and Harten, M. Abstr. *J. Allergy*, 9: 394, 1938.
4. Walzer, M., Gray, I., Straus, H. W. and Livingston, S. *J. Immunol.*, 34: 91, 1938.
5. Kaijser, R. *Arch. f. klin. Chir.*, 188: 36, 1937.
6. Afendulis, T. C. and Gullzow, M. *Ztschr. f. d. Exper. Med.*, 104: 167, 1938.

# SUCTION OPERATION FOR TOTAL REMOVAL OF CATARACT

## REPORT OF 228 CONSECUTIVE CASES

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THE writings of preceding and contemporary ophthalmic surgeons provide us with an immense reservoir of descriptive material and statistical fact which make a comparative study of the various surgical methods of cataract removal a task of huge proportions. Even the study of the development of a single technique is a work of no inconsiderable magnitude. And yet it is extremely important that the surgeon be kept fully and reliably informed regarding the different techniques available in order that he may evaluate in his own mind, and adapt to his own particular skill and ability, that method which will be most successful in his own hands. It seems inconsistent with good reason to insist, as many do, that the inexperienced surgeon, or one who operates only occasionally, should learn to use an old and perhaps inefficient technique simply because it has the approval of others.

There can be no doubt today of the superiority of intracapsular over extracapsular methods. Clear media with no need for repeated needling operations; lower incidence of postoperative inflammatory reactions; and avoidance of the depressing period of waiting for cataracts to ripen are all well recognized advantages of intracapsular methods.

That many general ideas regarding the Barraquer suction cataract operation[1] have never been adequately understood and that others pertain to an earlier and now outmoded stage in the evolution of the technique is not universally recognized. It is the earnest desire of the writer to call attention to a need for revision of these concepts. Unfortunately simple facts are often the most difficult to explain because they are frequently obscured by criticism based upon a lack of understanding and by the devious purposes of commercial interests desiring to introduce new instruments and equipment. It should also be recognized that even after a surgical technique has reached a stage of practical perfection it continues to undergo many minor personal modifications so that aside from its basic features the operation eventually becomes quite individualized among the surgeons who use it. Perhaps no two surgeons employing the suction technique do so in exactly the same way. It should be understood that this discussion pertains primarily to the technique as described below.

The vacuum or suction method for cataract extraction is not among the older and more traditional procedures. However, it has been widely enough used by outstanding European and American surgeons so that reliable statistics on large series of cases can be compared with those from the older methods of intracapsular and extracapsular extraction. The suction operation now stands as the first important and successful surgical cataract technique utilizing modern electrically driven precision instruments and embodying an original and radical departure from all of the older ideas and previous concepts regarding the surgical removal of cataracts.

Barraquer was first to describe the removal in toto of cataracts by the use of an electrically driven vacuum pump. The suction idea first occurred to him while watching a leech, which was stuck to the side of an aquarium, lift a small pebble with its caudal end. Barraquer devised the original

vacuum equipment and instruments which made possible the practical application of the method. With a thorough realization of the remarkable possibilities of his empirical efforts, he attempted at once to explain all the workings of the technique by his own concepts of its mechanics, but many of his theories are not now universally accepted. This is especially true of his idea of the importance of a pulsating or interrupted vacuum. The mechanics of this Barraquer attempted to explain by analogy to a weight jerked from the end of a piece of string. Few today would consider any value in the use of a pulsating or intermittent vacuum. A continuous and smoothly applied vacuum is considered preferable. Castroviejo[2] is emphatic in his opinion that a pulsating vacuum is quite unnecessary.

The vacuum may be controlled either by the tongue, by a foot valve or by the finger controlling an opening in the suction end piece. For over two years I employed the lingual vacuum control as devised by Nugent[3] but later returned to the use of the foot valve which I consider more convenient and sanitary; and which permits better control of the amount of vacuum desired. The optimum amount of vacuum varies according to the case. It must be applied gradually in order to allow the capsule and lens substance to mould into the cup. The manner of application and degree of vacuum are governed by individual judgment. In most cases a vacuum equivalent to 50 to 55 cm. of mercury is sufficient to rupture the zonule, but occasionally from 1 to 10 cm. more are required. Valves permit the application of the precise amount required. The valve is also a safety mechanism, for it permits the operator to release the vacuum instantly. Mechanical suction syringes do not allow for this variable control of the degree of vacuum nor for its immediate discontinuence.

Anesthesia, mydriasis, akinesis, lid control, retrobulbar injection and precaution against infection are accomplished in accordance with individual preference. They have been widely and extensively discussed in the literature and there is no need for lengthy discussion here.

For surface anesthesia the writer prefers a ½ of 1 per cent pontocaine solution because of its stability and because it does not have the dehydrating effect upon the corneal epithelium that a comparably effective solution of cocaine has.

For mydriasis Barraquer's solution containing 2 per cent euphthalmine is alternated with instillations of 1:1000 adrenalin. Several instillations produce rapid and adequate mydriasis of short duration. If constriction of the pupil is desired at the termination of the operative procedure, these mydriatics will not prevent the effective use of eserine as does mydriasis with atropine.

Before beginning the operation the lower lacrimal punctum is dilated and 1 to 2 c.c. of 1:3000 bichloride of mercury in petrolatum (White's ointment) is injected directly into the lacrymal sac. This has a twofold purpose: (1) it destroys any infectious organisms that may be lurking in the lacrimal passages; (2) it prevents the gastric upsets which frequently result from the toxic effects of excessive and rapid absorbtion by the nasal and pharyngeal mucosa of many of the solutions which drain through the lacrimal passages from the conjunctival sac.

Akinesis may be obtained either by the method of O'Brien[4] or by that of Van Lint.[5] Lid anesthesia is not important but it adds to the patient's comfort and may be easily attained by injecting a few c.c. of a solution of 2 per cent procaine with adrenalin into the subcutaneous tissues of the lids. Retrobulbar injection of 1.5 c.c. of the same solution produces a deep anesthesia of the entire globe and temporarily reduces the intraocular tension.

Lid control is most efficiently obtained by the method of Horner[6] which eliminates the work of an assistant. The sutures are placed in the lid at the beginning of the operation. A speculum is used only until the corneal section and iris incision have been made and the conjunctival sutures are

placed. The speculum is then removed before delivering the lens and thenceforth the lids are controlled by the sutures weighted

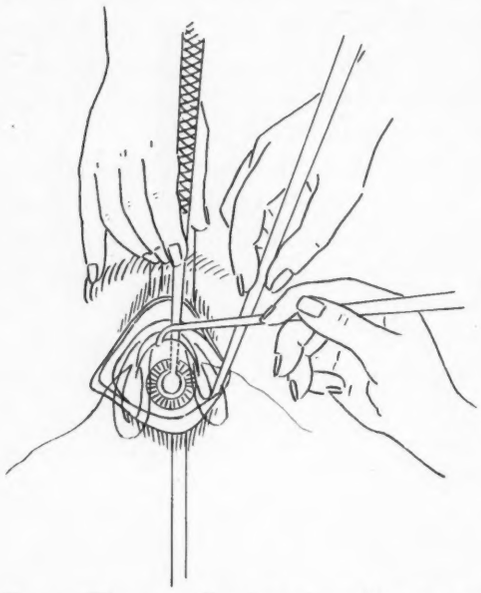

Fig. 1. Direct application of suction cup to anterior capsule. Corneal section held open with forceps by assistant. Slight counter-pressure with ball-tip forceps below.

with small hemostats. In this way, assistants' hands need not occupy any important part of the operative field and there is no need for a superior rectus suture.

An external canthotomy is performed when necessary.

The corneal section comprises slightly less than one-half the circumference of the cornea and includes a narrow conjunctival flap. A continuous or running conjunctival suture with attached atraumatic needles is placed in the conjunctival flap before the lens is delivered. The loops are drawn back into the canthi so that they do not interfere with the lens as it emerges from the wound. The ends of this suture extend beyond the eye so that by pulling them taut the wound may be closed immediately after delivery of the cataract. In this way, if any vitreous tends to prolapse, its loss may be kept to a minimum.

The ideal surgical and cosmetic result is attained with a round, active pupil and with the cataractous lens entirely removed. Incarceration of the iris in the wound is a complication to be avoided, especially when a total iridectomy has not been made. Both of these facts, as well as the probable behavior of the patient postoperatively should be borne in mind at the time the decision regarding the type of iris incision is made. In general, I prefer a peripheral iridotomy by Elschnig's[7] method, making a small slit near the iris root with a DeWecker scissors alone and avoiding the injury to the iris common when forceps are used. Where the iris has good tone and the slit-lamp reveals no slits or other signs of atrophy, where there is no posterior synechia, and where the iris is not injured in making the corneal incision, a peripheral iridotomy is the ideal way in which to deal with the iris. In aged or one-eyed patients, in patients who cannot be kept quiet because of some bodily infirmity, or where posterior synechia or other evidence of former uveal inflammation is present, where the pupil cannot be dilated, or in the presence of extensive uveal atrophy or chronic glaucoma, a narrow total iridectomy is preferred. Posterior synechia does not preclude the possibility of intracapsular cataract extraction as is sometimes stated. In this series of 228 cases, total iridectomies were performed in 148 cases. In the remaining eighty cases the iris was dealt with in such a manner as to leave the pupil round and intact at the completion of the operation. A preliminary iridectomy was done only for some one-eyed patients or in the presence of an associated glaucoma or a high persistent arterial hypertension.

To Barraquer the most important function of the suction end piece or erisiphake was to rupture the zonule. He even referred to this instrument as a pneumatic forceps and zonulotome. At first Barraquer advocated turning or tumbling the lens into the anterior chamber by rotating the erisiphake about the axis of its handle. Later he extracted the cataract without tumbling it and felt he had made distinct progress. Van Lint,[8] Fisher,[9] Green,[10] Wright,[11] Saint-Martin,[12] LaGrange[13] and others have described their methods of using suction in

which the tumbling maneuver is given considerable prominence. LaGrange, however, has recently emphasized the importance of first rupturing the zonule by rotating the lens in the plane of its equator before tumbling it. The version or tumbling maneuver is primarily intended to complete the rupture of the zonule. If this can be accomplished by rotating the lens by a tortional movement, the need for tumbling is abolished and the cataract can be delivered directly by traction and slight counter-pressure without ever turning the end of the suction end piece out of view. In this way the forensic objection to the suction technique—that there exists an imminent danger of aspirating the vitreous should the lens become detached from the vacuum cup—is entirely obviated.

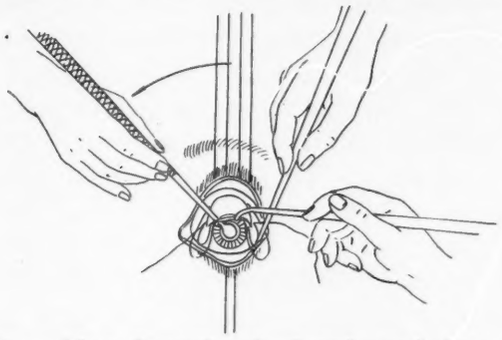

Fig. 2. Note change in axis of suction end piece as the entire lens is rotated around the axis, passing through anterior and posterior poles of crystalline lens.

The author's method of direct extraction, without tumbling, by rupturing the zonule by rotating the lens in the plane of its equator, and by keeping the open end of the suction end piece constantly in view is illustrated in Figures 1 to 4. It is particularly emphasized that the incidence of vitreous loss is reduced to a minimum, seldom attained with any other technique.

In this series of 228 cases, in previous studies[14] and in an extensive experience with hundreds of other suction operations I have never observed the loss of a single drop of vitreous by aspiration into the suction end piece. When vitreous loss occurs with this technique it is due to excessive counter-pressure, to movement by the patient, to contraction of the orbicularis muscles, or other undue pressure on the globe by instruments or by the lids. The vitreous escapes into the conjunctival sac just as in any forceps operation and does not disappear into the suction apparatus.

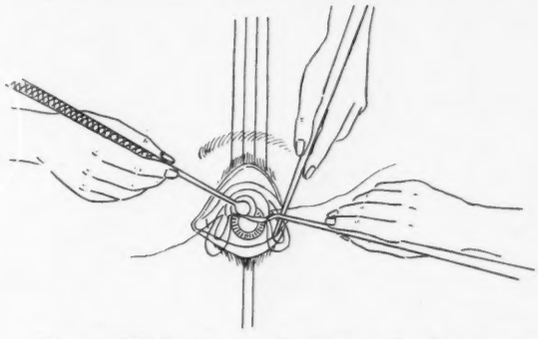

Fig. 3. With the zonule ruptured, the cataract is delivered *by traction* and slight counter-pressure.

Considering the anatomy of the eye and the physical problem involved in the direct extraction of cataract by the utilization of a pneumatic forceps, it is not surprising that vitreous loss is neither an imminent danger nor an important complication. The crystalline lens is suspended by the zonular fibers which extend from the anterior and posterior capsule of the lens in the region of the equator to the ciliary valleys and processes of the ciliary body. These zonular fibers form an open network through which aqueous filters and also affords a direct open pathway between the retrolental space and the posterior chamber. The posterior chamber and the anterior chamber are directly continuous through the pupil. In other words, after the corneal section has been done and the eyeball opened, atmospheric pressure is transmitted directly through the pupil and the interspaces between the zonular fibers to the anterior condensation layer of the vitreous. Therefore when the vacuum cup is placed on the anterior capsule of the lens with the wound held open, the vacuum is not transmitted to the vitreous. Atmospheric pressure is not removed from the anterior vitreous surface nor from the posterior surface of the lens.

Failure to comprehend these simple facts has led writers to describe the "pushing

out"[15] of the lens by the vitreous as a vacuum cup is applied to a part of the anterior lens surface. This is an entirely

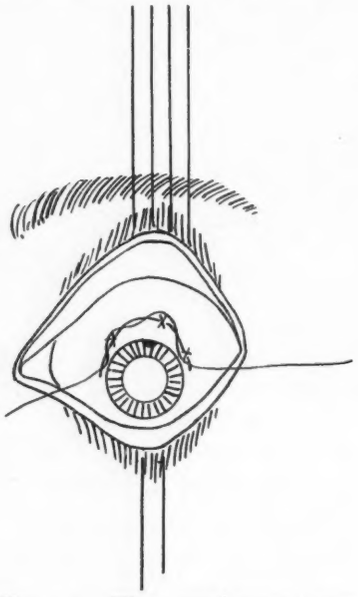

FIG. 4. The continuous conjunctival suture is pulled taut, closing the wound immediately after delivery of the cataract.

false concept of the mechanism of the suction technique. (See Figs. 1–4.) When the proper degree of vacuum has been applied, the lens can be lifted from the anterior vitreous surface exactly as with any other type of forceps but with much less danger of rupturing the capsule. In a recent paper[16] Barraquer complains that much of the criticism directed toward the suction operation has been based on incorrectly executed operations, diversified techniques, and a complete misunderstanding of the essential factors. He points out that one should be especially careful not to attribute to a technique disadvantages which should be charged to the surgeon.

A peripheral iridotomy or an iridectomy makes an even more direct pathway for the application of atmospheric pressure onto the anterior vitreous surface.

Clinical records of 228 consecutive cases recently operated on by the author by the technique described afford an interesting statistical study. The patients ranged in age from 19 to 94 years. In over 80 per cent of the cases the cataracts were in incipient stages. Inability or failure to deliver the cataract in toto occurred in ten cases, and cataracts were totally removed in slightly more than 95 per cent. These percentages are in close agreement with those recently reported by Arruga.[17] Vitreous loss occurred in only thirteen cases, or slightly less than 6 per cent. The amount varied from a few beads to a moderately large amount, but in no case did loss of vitreous prevent healing or recovery. Postoperative wound infection occurred in one case in the entire series.

It should be generally understood that 100 per cent corrected visual acuity is obtainable in every case operated by an intracapsular technique without complications, providing that the parts of the visual apparatus other than the lens are normal. However, since in many cases ocular structures other than the crystalline lens have undergone pathologic changes, normal visual acuity is not to be expected always. Statistics regarding visual results are therefore of little value unless accompanied by extensive and modifying pathologic descriptions. All that is necessary is that each patient be given an accurate appraisal of the condition of his eyes before operation and an estimate of how much visual improvement may be expected.

## SUMMARY AND CONCLUSIONS

In total extraction of cataract the suction operation is highly successful, permitting intracapsular extraction in over 95 per cent of the cases in which it is attempted. Vitreous loss is not a common or serious complication.

On the combined basis of complete removal of the cataract, minimal complications, and best possible visual results, the conclusion is reached that the suction operation stands out among the best surgical techniques for removal of cataracts.

## REFERENCES

1. BARRAQUER, I. Phacoerisis. *Arch. Ophth.*, 50: 307, 1921; 51: 448, 1922.
BARRAQUER, I. In Fisher. Senile Cataract.[9]

2. Castroviejo, R. Personal communication.
3. Nugent, O. *Tr. Sect. Opbtb., A. M. A.*, p. 307, 1933. Nugent and Fisher. *Illinois M. J.*, 64: 320, 1933. Nugent, O. *Texas State J. Med.*, 32: 664, 1937.
4. O'Brien, C. S. Local anesthesia in ophthalmic surgery. *T. Sect. Opbtb., A. M. A.*, p. 237, 1937.
5. Van Lint, A. Paralysie palpébral temporaire provoqué dans l'opération de la cataracte. *Ann. d'ocul.*, 151: 420, 1914.
6. Horner, W. D. Suture of lid control in cataract operations. *Am. J. Opbtb.*, 18: 33, 1935.
7. Elschnig, A. Simple senile cataract extraction with incision of the root of the iris. *Ann. Opbtb.*, 21: 447, 1912.
8. Van Lint, A. Extraction latérale intracapsulaire de la cataracte. *Arcb. d'opbtb.*, 43: 591, 1926.
Van Lint, A. In Fisher, A. Senile Cataract.[9]
9. Fisher, W. A. Senile Cataract. Chicago, 1937. Adair Printing Co.
10. Green, A. S. *Tr. Sect. Opbtb., A. M. A.*, p. 313, 1917.
Green, A. S. Intracapsular expression extraction of cataract. *Arcb. Opbtb.*, 51: 338, 1922.
11. Wright, R. *Tr. Opbtb. Soc. United Kingdom*, vol. 47, 1927.
12. DeSaint-Martin, R. Traité d'ophthalmologie, vol. 8. Paris, Masson. In press.
13. LaGrange, H. L'extraction pneumatique de cristallin par rupture zonulaire. *Ann. d'ocul.*, 174: 387, 1937.
14. Blaess, M. J. Barbiturate therapy and cataract surgery in Parkinsonism. *J. Iowa State M. S.*, Nov., 1937.
Blaess, M. J. Dinitrophenol cataracts—removal by Barraquer intracapsular operation. *Eye, Ear, Nose and Throat Monthly*, Nov., 1938.
Blaess, M. J. Circumcorneal telangiectasis. *Arcb. Opbtb.*, 21: 1031, 1939.
15. Dimitry, T. J. Evolution of a sucking disc for intracapsular extraction of cataract. *Arcb. Opbtb.*, 21: 261, 1939.
16. Barraquer, I. La suture cornéo-sclérale dans l'extraction totale de la cataracte sénile. *Ann. d'ocul.*, 176: 470, 1939.
17. Arruga, H. Le pourcentage d'extractions totales dans l'opération de la cataracte. *Ann. d'ocul.*, 176: 300, 1939.

# POSTOPERATIVE PULMONARY COMPLICATIONS

## AN ANALYSIS OF ETIOLOGY, DIAGNOSIS, PROPHYLAXIS AND TREATMENT

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THE subject of pulmonary complications following surgical procedures has long been a source of worry to both surgeons and internists. The incidence is reported as high as 5 per cent in all operations, and 10 per cent in those limited to the abdomen. A University of Pennsylvania study[1] shows a morbidity of 1.68 per cent and a mortality of 0.54 per cent, with 8.35 per cent of the operative mortality entirely attributable to the pulmonary lesion.

### ETIOLOGY

No single factor is believed responsible, but certain factors related to the incidence and etiology should be stressed.

1. *Site of Operation.* The highest incidence is found after abdominal surgery, especially in the upper abdomen. King[2] found an incidence of 14 per cent among 3,000 abdominal cases (hernioplasty included), and only 1 per cent among 4,000 operations elsewhere on the body, thyroidectomy comprising the major portion of this latter group. It is an axiom that *the more closely the operative procedure approaches the diaphragm, the greater is the incidence of postoperative pulmonary complication.* This is explained by resultant immobilization of the abdominal muscles together with elevation of the diaphragm, often as much as 6 to 8 cm., with a corresponding reduction in vital capacity. A 50 per cent reduction in vital capacity after lower abdominal operations and 85 per cent reduction after gall-bladder operations were noted by Churchill and McNeil.[3] These changes reach their height twenty-four hours after operation, and return gradually to the preoperative level over a period of one to three weeks. Davis[4] observed that the preoperative chest roentgenogram in the expiratory position is almost identical with the postoperative roentgenogram in the inspiratory position.

2. *Sepsis in Operative Field.* Upper abdominal sepsis undoubtedly adds to the restriction of diaphragmatic contraction. Ryan[5] stresses the importance of infection in the operative field in the production of postoperative pulmonary complications. The manner of spread may be by direct extension, via lymphatic channels, or by embolic means.

3. *Preëxisting Respiratory Infection.* Acute infections are quickly recognized and unequivocally forbid all but emergency operations. Subacute and chronic infections are too often glossed over in the preoperative physical examination; they demand equal consideration. Acute respiratory infections are often acquired during the preoperative period of study, possibly due to unaccustomed changes in dress, bed coverings, and ventilation, or to transportation to and from surgery along drafty corridors.

4. *Anesthesia.* Endless controversies have arisen as to the part inhalation anesthesia plays as a causative factor. Irritative inhalation anesthetics, with resultant increase in bronchial secretions and formation of mechanical plugs, are potent factors. But, according to Brock,[6] "It is not sufficiently appreciated that the incidence of a chest complication depends on a combination of several circumstances and but rarely on one factor alone. To imagine that the simple replacement of inhalation anesthesia by local or spinal anesthesia for a patient known to be a bad risk will avoid such a complication shows a childlike faith born of

inexperience or insufficient observation. While it is desirable to avoid an irritant inhalant, it is even more important to take certain other precautions, and, unless these be taken, it is absurd to be disappointed with the results of the use of local or spinal anesthesia." Brown and Debenham[7] report an incidence 4.29 times greater with spinal than with inhalation anesthesia.

5. *Bronchial Obstruction.* Several factors combine to promote thoracic immobility and shallow breathing, with the subsequent formation of bronchial mucus plugs. Tracheobronchial secretions, increased by inhalation anesthesia or preëxisting bronchial infection, accumulate dependently during a prolonged operation in the supine position. Muscle splinting following abdominal incisions and large dosages of opiates promote such an accumulation. In many cases this accumulation is disposed of during recovery as soon as the patient vomits or coughing is reëstablished. In others a mucus plug is formed. Constricting bandages, often made tighter by gaseous distention, hinder expulsion of such a plug. Retention is also facilitated by immobility, whether due to senility, obesity, primary causal illness, prolonged, exhaustive operation, lack of normal stamina, or to the condition of major invalidism often urged upon the patient by his relatives and friends.

Tracheobronchial secretions are raised largely by the "tussic squeeze" or "bechic blast." This cough is a protective mechanism, stimulated by the accumulation of secretions which it attempts to raise. Prescription of cough sedatives to relieve such a cough cannot be too strongly condemned.

It is doubtful whether atropine should be used, either pre- or postoperatively, as it undoubtedly hinders drainage by thickening secretions. It is better to aspirate secretions as they arise.

Band and Hall[8] have shown by experiments on dogs that the three factors that tend to cause massive atelectasis are the presence of a plug of tenacious mucus in a bronchus, the abolition of the cough reflex (as after morphia), and limitation of respiratory movements. Coryllos[9] analyzes various theories as to the etiology of pulmonary collapse, and concludes, "I consider bronchial obstruction as the only *determining* cause of atelectasis."

6. *Predisposing Factors.* The highest incidence is during middle life. It is twice as high in men, possibly due to their greater reliance on abdominal respiration, increased exposure to the elements, and more rigid costal cartilages. A higher percentage of upper abdominal surgical lesions, with concomitant postoperative splinting of the diaphragm, raises the incidence in males. The seasons of prevalent upper respiratory infections have also the highest incidence of pulmonary complications.

## CLASSIFICATION

Postoperative pulmonary complications may be classified according to their time of onset.

1. *First to Fifth Day.* Atelectasis, of the obstructive type, due invariably to a plugged bronchus; rarely occurs after the fifth day.

2. *Fifth to Eighth Day.* Unclassified group, in which pneumonia apparently falls; preceded by atelectasis.

3. *Subsequent to Eighth Day.* Embolic complications and lung abscess, of which atelectasis is the precursor.

Such a scheme is not meant to include every case, but its adoption facilitates early and often life-saving therapy.

1. *Atelectasis.* This is a frequently met complication which arises within a few hours to five days after operation, usually occurring in a quiet patient suffering intensely from pain. Many varieties of atelectasis are recognized, varying from massive or lobar collapse, with clear-cut physical signs, to a patchy or lobular, scattered atelectasis with signs and symptoms often erroneously labeled as bronchitis or bronchopneumonia. However, lobular atelectasis is often the forerunner of these complications. The condition is frequently bilateral.

The onset is almost always abrupt, with

rapid and shallow respirations; occasionally there is pain and distress. The temperature is elevated, and the pulse rises to 120 to 160. As the symptoms progress, cyanosis rapidly intervenes, and dyspnea, fatigue, apprehension, sweating, postural inclination towards the affected side, and a sense of oppression or retrosternal discomfort are noted. A typical fruity cough, from "gargling" of retained, thick, mucopurulent material in the trachea and large bronchi, is quite characteristic.

Most of the complications occur on the same side as the operation. This is probably not because of the operative site, but occurs as a result of the lesser angle of the right stem bronchus with the trachea, so that the tracheal secretions gravitate into the right lung, analogous to foreign bodies.

The diagnostic physical signs are, displacement of the heart to the affected side, lagging or decreased expansion with an impaired to dull percussion note, and breath sounds which are strikingly variable, depending on expectoration of mucus plugs; loud bronchial breathing may appear in a previously silent area after expectoration of thick sputum.

Roentgenograms in the diagnosis of atelectasis are unquestionably of great value, but they are not always typical, and usually are interpreted as pneumonia. The elevation of the diaphragm and narrowing of the interspaces on the affected side, together with displaced organs, are the most striking diagnostic features of the roentgenogram. These findings are absent in partial atelectasis, and it is almost impossible to differentiate these cases from pneumonia by *x*-ray or physical examination. The author believes they should be diagnosed and treated as atelectasis if they occur within the first five days after operation.

The sputum is typically thick, viscid, mucopurulent, tenacious, and not blood-stained, in contrast to the rusty sputum of a true pneumonia or the blood-tinged sputum of a pulmonary infarct.

A postoperative pulmonary complication having its onset within five days of operation should be considered atelectasis until otherwise excluded. Pneumonia is believed to be a further stage of the same atelectatic process. Lung abscess follows the pneumonitis which was superimposed upon the atelectasis. Pol Coryllos[9] considers atelectasis an initial, integral part of postoperative pneumonia; he believes the latter to be identical with pneumococcic atelectasis. Bronchial obstruction, according to the same author, is the starting point of postoperative bronchitis, lobar or lobular pneumonitis, and probably of abscess and gangrene. The particular condition arising depends upon the virulence of the infecting agent.

An early diagnosis is therefore essential so that treatment may not be delayed. The longer the interval between onset of symptoms and the beginning of treatment, the more danger is there of pulmonary abscess. The prognosis of properly treated cases is good, recovery occurring in some cases as a result of one treatment (therapeutic bronchoscopy).

2. *Pneumonia.* True lobar pneumonia is a rare sequel to operation. Postoperative bronchopneumonia is more frequent and is almost always a very serious condition, occurring as it does in a type of patient most unfitted to withstand a heavy infection. In the past it has been commonly diagnosed in error when only atelectasis was present; it actually represents an advanced stage of atelectasis. Prophylaxis, therefore, must be energetically directed towards the initial atelectatic process. Once postoperative bronchopneumonia has developed, the clinical picture resembles the medical entity.

3. *Lung Abscess.* Lung abscess is always a late complication, often delayed as long as two weeks after operation. The atelectatic processes progress into a pneumonitis and then form an abscess cavity. The diagnosis is based on a rapid progression of atelectatic symptoms, the onset of cough with sputum, gradually becoming more severe, and the sudden expectoration of large amounts of very foul sputum (due to the rupture of the abscess into a bronchus). The prognosis varies directly with the time

of institution of therapy and the time of onset of the disease.

4. *Embolism and Infarction.* This occurs in most cases after the eighth postoperative day, after physical exertion, in a patient without pain, as contrasted to the early onset of atelectasis in a quiet patient with pain. There is usually no collapse except with a massive embolus. Recognition is based on sudden pleuritic pain below the scapula, slight elevation of temperature, and tachypnea, with development two to three days later of friction rub in 50 per cent of cases,[9] dullness, and either a nonproductive cough or blood-tinged, frothy sputum.

5. *Bronchitis.* Bronchitis occurs in about 3 per cent of cases, as a simple productive cough with fever and no abnormal signs in the chest beyond a few râles. Resolution follows in a short time with no evidence of severe or permanent damage. The danger lies in the application of this simple diagnosis to more serious conditions, which it may precede.

## PROPHYLAXIS

Postoperative pulmonary complications may largely be prevented if certain measures suggested by their etiology are utilized in the care of the patient.

1. *Preoperative Care of the Respiratory Tract.* Only the most urgent operations should be done in the presence of an acute upper respiratory infection. Chronic disease, such as tonsillar and dental sepsis, should be eliminated before all elective surgery; pulmonary abscess is not infrequently the result of infected teeth. Impatience of patients and relatives should not prevent postponement of such cases. Patients should be seen on the morning of operation, and examined for recently acquired colds or changes in temperature.

2. *The Operation.* An incision as atraumatic as possible, causing the least afterpain, and the least splinting of the upper abdomen and diaphragm, will prevent in part postoperative hypoventilation and its sequelae. Careful handling of tissues will result in less shock, with its concomitant immobility and lowering of resistance. Jones and McClure[10] advocate a transverse incision for upper abdominal operations, reporting only five cases of embolism with two deaths and no cases of atelectasis or pneumonia in 125 surgical cases in which this incision was employed.

Extremely tight upper abdominal dressings are to be avoided, and, unless there is a definite indication, upper abdominal binders are not to be used. Adhesive strips placed in "X" fashion in epigastric dressings should prevent constriction of the lower ribs.

3. *Anesthesia.* An irritant inhalation anesthetic is to be avoided whenever possible, and if one is given, the anesthesia should be smoothly and skillfully maintained. The reduction of the anesthetic period to the shortest possible time is of definite prophylactic value. Every patient who presents himself must be *individualized* and the anesthetic selected that is safest from every standpoint.

Pol Coryllos[11] has suggested the use of intratracheal insufflation anesthesia with bronchial suction, thereby preserving respiratory potency and pulmonary ventilation, and facilitating elimination of bronchial contents before, during, and after operation. Such anesthesia is becoming more popular as skill in administering it is more widely acquired.

4. *Morphia and Atropine.* Opiates should be used postoperatively to lessen sensibility to pain, but in doses small enough not to inhibit respiration or the cough reflex; this will allow bronchial drainage by cough without fear of wound pain. Pantopon may be substituted for morphia because it has a noticeably decreased tendency to thicken bronchial secretions and thereby causes less interference with their drainage. Belladonna or atropine given postoperatively first suppresses secretion, but then renders it more viscid and hinders expulsion.[9]

5. *Position and General Care.* The patient should be elevated to semi-Fowler position immediately after he reacts from the anesthetic; if he has received spinal

anesthesia, the foot of his bed should be elevated 6 inches for two hours, then placed flat for two hours, and then low Fowler's position. The benefits are derived from less hypostatic congestion and more complete lung expansion.

Coughing, to rid the tracheobronchial tree of sputum, should be encouraged by abdominal support and judicious use of narcotics. Supervised deep breathing exercises, performed with slow inspiration (less pain) and rapid expiration (to raise mucus to cough level), should be conducted at frequent intervals as soon as consciousness is regained. The patient should be turned from side to side after abdominal operation, at intervals of one-half to two hours (varying with quantity of bronchial secretion), when operative conditions safely permit it. Bronchial drainage should be insured for at least forty-eight hours postoperatively, when the lung defenses are impaired.

6. *Carbon Dioxide Inhalations.* Eliason and McLaughlin,[1] Lahey,[12] and MacKenzie[7] endorse the routine use of carbon dioxide inhalations, noting a decreased incidence of pulmonary complications in patients receiving the benefit of its use. However, King[2] and Ryan[5] report no appreciable differences in two large controlled series. The hyperventilation, maintenance of the inspiratory position, and violent tracheobronchial movements resulting from the use of carbon dioxide inhalations open atelectatic areas and aid the expulsion of obstructive secretions retained from hypoventilation. Carlson[13] believes this effect to be too temporary to be of value, as he found shallow abdominal respirations resumed within two to five minutes after discontinuance of the inhalations. Coryllos[9] claims that carbon dioxide inhalations decrease the hydrogen ion concentration of the exudate and thereby inhibit the growth of pneumococci, and also favor proteolysis of fibrin in the exudate, rendering it more expectorable.

As there are no untoward effects except diuresis and increased acidity of the urine, the inhalations should be given after all abdominal surgery, in a 10 to 25 per cent mixture with oxygen, for three minute periods at intervals of fifteen to thirty minutes; they should be begun immediately after operation and continued for four to six hours, or longer if deemed necessary. Hyperventilation at the conclusion of the operation should be a routine part of the anesthesia. However, "To expect to solve our problem by directing a nurse to hold a funnel over a patient's face at regular intervals is to put a very low value on the difficulties before us."[6]

7. *Drugs for Prevention of Embolism.* Various drugs have been advocated, principally by European investigators, for prophylactic use in reducing the incidence of postoperative embolism and infarction. The merits of these drugs—calcium gluconate, eupaverine, ephedrine, synephrin, and others—have not been substantiated, and they are seldom recommended. Peripheral thrombectomy has been successfully employed on some occasions as prophylaxis against embolism.

Patients with low basal metabolic rates may be given thyroid extract pre- and postoperatively, to raise metabolism and increase the rate of circulation.[14] Where the basal metabolic rate is low, the results have been favorable.

Eupaverine and papaverine are frequently used in treatment of cases of embolism which do not have a sudden termination.

Thus, no one factor is responsible for postoperative pulmonary complications, and, unless steps are taken to control every factor, no marked success is likely to be attained in their prevention.

## TREATMENT

If proper pre- and postoperative care is used, atelectasis will seldom occur, and pneumonia and lung abscess will thereby be prevented.

1. *Atelectasis.* Once atelectasis is established, treatment should be directed to overcome bronchial occlusion and insure

free drainage of the bronchial tract. Adjustment of tight dressings, changes in posture, deep breathing, active efforts at coughing, and 10 to 25 per cent carbon dioxide inhalations are of inestimable value. Postural drainage, performed by having the patient breathe deeply and cough while lying in the lateral position with the involved side uppermost, will often expel much obstructive mucus. The patient should lie in bed on the non-involved side, to facilitate drainage and expansion of the atelectatic areas. If these routine procedures do not result in either preventing or relieving the atelectasis, further treatment is imperative. Bronchoscopy is indicated where the patient cannot expectorate or is growing worse.[4,9] The fact that a patient is practically moribund does not contraindicate a bronchoscopy. A carefully planned bronchoscopy can easily be done in any hospital, at which time the pulmonary secretions are aspirated, and inspissated plugs of mucus, if present, can be removed.

The routine administration of oxygen to a patient with a postoperative pulmonary complication, especially one with a plugged bronchus, cannot be too strongly condemned. Remember, the plug must be removed before oxygen can reach the alveoli.

2. *Pneumonia.* Postoperative pneumonia should never develop if proper and immediate treatment of the preceding atelectasis is instituted. Once it has developed, it demands the same treatment as any other pneumonia.

3. *Lung Abscess.* Lung abscess following an abdominal operation is primarily an atelectasis. Suppuration develops slowly, and may be prevented by bronchoscopic aspiration during the period of atelectasis. Of Davis'[4] fifteen cases of postoperative atelectasis which did not respond to the ordinary palliative procedures and had prompt bronchoscopic aspiration of the secretions, not one developed an abscess. Chevalier Jackson[15] has reported similar results. Early bronchoscopic aspiration with direct medication cleared up several of Negus'[16] cases of lung abscess.

Atelectasis is not the initial pathologic state in abscesses following operations about the upper air passages. In these cases, there is a fulminating onset and rapid abscess cavity formation as the initial lesion.

4. *Embolism and Infarction.* Embolectomy has been successfully performed in opportune cases, but a rapidly fatal course forestalls therapy in many instances. Eupaverine and papaverine are of distinct aid in favorable cases.

5. *Bronchitis.* The therapy of uncomplicated cases is purely medical, as for any other bronchitis. Bronchopneumonia must be guarded against, and appropriately treated when it arises.

The following two case histories are presented in illustration of the above principles.*

Case 1. D. McG., 21 year old white American youth, in previous good health, was admitted to Emergency Hospital, Washington, D. C., on the evening of January 8, 1938 with a two day history of abdominal pain. Under a smoothly administered avertin-ethylene anesthesia, a McBurney incision revealed a ruptured appendix with localized peritonitis; appendectomy and peritoneal drainage were performed, and the patient returned to his room in favorable condition. Progress was satisfactory until the early morning of January 11, when, with dramatic suddenness, the patient was aroused from his comfortable state by a sudden fit of painful coughing and labored breathing. Cyanosis rapidly became prominent and his pulse could not be obtained by the nurse. Cardiorespiratory stimulants were administered and the patient was placed in an oxygen tent, with a prompt general improvement of his condition. The immediate temperature of 106°F. (rectal) and pulse of 154 soon fell to 101 degrees and 130, respectively, and remained there. Respirations varied from 36 to 44 per minute. Morphia, parenteral fluids, and an oxygen tent, required without intermission, sustained the patient for the next two days. Portable *x*-ray and repeated physical examination at this time led to a diagnosis of massive pneumonic processes, involving the entire right lung and one-third of

* From the private practice of Dr. Edgar Davis, Washington, D. C., whose constant help and advice made this study possible.

the left lung, attributed with question to an aspiration process.

Increasing cyanosis, a rising temperature, pulse, and respiratory rate, and progressive irrationality necessitated a therapeutic bronchoscopy on the evening of the 13th, and 65 c.c. of thick, tenacious, mucopurulent secretions was aspirated from the larynx and both stem bronchi. The inflamed mucosa was shrunken with cocaine-adrenalin solution, leaving both stem bronchi perfectly clear. Upon removal of the bronchoscope the patient volunteered that he felt tremendously improved and that he could breathe much easier. The oxygen tent was discontinued and was never required thereafter. The patient's color improved and remained good throughout the rest of his hospital stay. Roentgen examination thirty-six hours later revealed an absence of all consolidation and only a slight residual infiltration. The temperature dramatically fell to normal by the next morning and remained normal. Pulse and respiration gradually declined to a normal level over a period of four days. Convalescence was rapid and uneventful, and the patient was discharged in ambulatory status on January 22, eight days after bronchoscopy, and fourteen days after admission. Convalescence continued uninterrupted while at home.

*Comment.* Evidently this patient was suffering from a postoperative, bilateral, partial atelectasis, due to plugged bronchi. The onset was early and followed a septic abdominal operation with inadequate emergency preparation. Roentgenograms were misinterpreted and proper treatment was delayed sixty hours. The therapeutic value of bronchoscopic aspiration was dramatically illustrated.

Case 11. A. M., 45 year old Italian chef, male, in previous good health, was admitted to Garfield Hospital, Washington, D. C., on November 20, 1939 with the complaint of a small, incomplete, left inguinal hernia, the result of straining three months before. Physical examination and urinalysis were unremarkable. The next morning a hernioplasty was performed without difficulty under a smoothly administered ethylene anesthesia. The patient reacted favorably and voided, but began to cough lightly. The cough became progressively worse, with no expectoration, and he complained of growing chest pain. Twenty-four hours after operation he had marked respiratory distress with a chill; he became extremely apprehensive and felt as if he was "suffocating."

On examination, his temperature was 103°F., pulse 140, and respirations 34 per minute. There was some cyanosis of the face and lips, inspiratory dilatation of the alae nasi, and a productive cough. Chest examination revealed the trachea in the midline, no cardiac shift, dullness over the posterior right lung from the scapular spine to the base, with bronchovesicular breath sounds, many subcrepitant râles, and absence of whispering pectoriloquy and bronchophony. A blood count showed 11,400 leucocytes with 74 per cent polymorphonuclears and 5 per cent band forms, 19 per cent lymphocytes, and 2 per cent mononuclears. The sputum contained no type specific pneumococcus, and was thick, tenacious, and blood-tinged.

A diagnosis of postoperative partial atelectasis was made clinically, but a portable chest roentgenogram was reported as a pneumonic process in the lower right lung, despite some narrowing of the ribs and a slight shift of the heart towards the affected side.

The patient was placed on his left side, given no opiates or cough sedatives, and encouraged by his nurse to cough. When this failed to produce bronchial drainage after eight hours, and his condition grew steadily worse, he was bronchoscoped and several thick mucus plugs removed from the right stem bronchi. Immediately afterwards he coughed up several ounces of thick, blood-tinged sputum, and showed immediate improvement. Expectoration continued freely; respirations became easier, and his temperature, pulse, and respirations rapidly fell overnight to 99 degrees, 110, and 30 respectively. Twelve hours after bronchoscopy the chest signs had almost totally disappeared, but he continued to expectorate thick sputum in small amounts for the next forty-eight hours. During this time he received no cough sedatives or opiates, and was encouraged to cough in accordance with a written order. His temperature, pulse, and respirations continued to be normal, and symptomatically he complained only of a mild headache. The remainder of his hospital course was uneventful.

*Comment.* This is another case of atelectasis due to bronchial obstruction, this time

in an aseptic, uncomplicated case. The diagnosis was promptly made by an aware clinician, despite misinterpreted roentgenograms. Early bronchoscopic aspiration removed obstructing mucus plugs and facilitated bronchial drainage by other measures, resulting in a quick resolution of the atelectatic condition.

## SUMMARY

Because of prevalent misconceptions regarding postoperative pulmonary complications, an effort has been made to stress those salient factors in their etiology and diagnosis which will lead to more effective prophylaxis and treatment. It is the author's opinion that atelectasis is the primary pathologic process, and that pneumonia and lung abscess represent advanced stages of the initial atelectasis. Therefore, the time of onset is of major importance in diagnosis. Bronchial obstruction is the sole determining cause of postoperative atelectasis. The quantity and character of the tracheobronchial secretions, and the forces available for expulsion of these secretions, are the factors concerned in the formation of bronchial plugs. Any agent causing an unfavorable change in either factor may precipitate bronchial occlusion. Most often it is the result of an increased amount of thickened secretions with lessened ability to expel them. If infection is superimposed upon the atelectasis, pneumonitis results. Abscess formation follows if the organism is sufficiently virulent and proper treatment is delayed.

The following factors, involving the tracheobronchial secretions, powers of expulsion of the secretions, and pulmonary infection, have been discussed and evaluated: operative site, operative sepsis, acute and chronic preëxisting respiratory infection, dental sepsis, anesthesia, incisional trauma with resultant muscle-splinting, constricting dressings, with diaphragmatic limitation, immobility, use of morphia, atropine, and cough sedatives, and the predisposing factors of age, sex, and season.

The time of onset has been stressed in diagnosis. Complications appearing in the first five postoperative days are usually atelectasis. Pneumonia seldom occurs prior to the fifth day and most often makes its appearance between the fifth and eighth days. Embolic complications and lung abscesses are usually met after the eighth day. This differentiation is important in the diagnosis of atelectasis from bronchopneumonia, as the clinical picture and even the roentgenogram are often deceptive.

Prophylaxis must be directed towards the prevention of bronchial obstruction and pulmonary infection. Each of the several etiologic factors must be given serious consideration as to its potentiality. The following prophylactic measures have been recommended: preoperative care of the respiratory tract, including the prevention of "colds" after admission; incision and operation as atraumatic as possible; avoidance of tight upper abdominal dressings; an *individualized* anesthetic; judicious use of opiates with preservation of the cough reflex and elimination of pre- and postoperative atropine; semi-Fowler position, supervised deep breathing exercises, and frequent turning after consciousness is regained; encouragement to remove bronchial secretions by cough; carbon dioxide inhalations routinely after abdominal operations; and thyroid extract where the basal metabolic rate is definitely below normal, to prevent embolism.

Only the treatment of atelectasis is presented, for if this condition is treated promptly and efficiently pneumonia and lung abscess will not occur. Mention is made of therapy of embolism. Treatment of atelectasis means removal of the obstruction. Measures used in prophylaxis are also utilized in treatment. In addition, postural drainage, with the aid of cough while in the most favorable position for drainage, is recommended. Bronchoscopic aspiration is strongly recommended when the more conservative methods of drainage fail. Two illustrative cases are presented.

## CONCLUSIONS

1. No one factor is believed responsible for postoperative pulmonary complications. Bronchial obstruction due to a mucus plug and respiratory infection are the most potent factors.

2. Atelectasis, due invariably to a bronchus plugged with mucus, is the basis of most of these complications. Pneumonia and lung abscess represent advanced stages of a primary atelectatic process.

3. Diagnosis in most cases is difficult; roentgenograms are often misinterpreted, and clinical findings are variable. Erroneous diagnosis of pneumonia where only atelectasis exists is frequent. The time of onset is a major consideration in the differential diagnosis.

4. Bronchial drainage, by cough, posture, or bronchoscopy, is essential if atelectasis and its more serious sequelae are to be prevented.

5. Bronchoscopy is a safe procedure in well-trained hands. Its early use when indicated will prevent formation of most lung abscesses.

6. Prophylaxis must not be based on any *single* etiologic factor; the patient must be considered from *every* potentiality.

## REFERENCES


1. Eliason, E. L. and McLaughlin, C. Postoperative pulmonary complications. *Surg., Gynec. & Obst.*, 55: 716, 1932.
2. King, D. S. Postoperative pulmonary complications; a statistical study based on two years personal observation. *Surg., Gynec. & Obst.*, 56: 43, 1933.
3. Churchill, E. D., and McNeil, D. Reduction in vital capacity following operation. *Surg., Gynec. & Obst.*, 44: 483, 1927.
4. Davis, E. Personal communication.
5. Ryan, T. J. Hyperventilation in abdominal surgery. *J. A. M. A.*, 107: 267, 1936.
6. *Brock*, R. C. Postoperative chest complications: a clinical study. *Guy's Hosp. Rep.*, 86: 191, 1936.
7. MacKenzie, J. R. $CO_2$ in prophylaxis and cure of postanesthetic respiratory complications. *Brit. M. J.*, 3716: 561, 1932.
8. Band, D., and Hall, I. S. Massive collapse of the lungs. *Brit. J. Surg.*, 19: 387, 1932.
9. Coryllos, Pol N. Postoperative pulmonary complications and bronchial obstruction. *Surg., Gynec. & Obst.*, 50: 795, 1930.
10. Jones, D. F., and McClure, W. L. Influence of transverse upper abdominal incision on incidence of postoperative pulmonary complications. *Surg., Gynec. & Obst.*, 51: 208, 1930.
11. Coryllos, Pol N. Etiology, prevention, and treatment of postoperative hemorespiratory complications in surgical treatment of tuberculosis: endotracheal anesthesia combined with bronchial suction; 84 cases—152 operations. *J. Thoracic Surg.*, 2: 384,
12. Lahey, F. H. Management of some complications following abdominal operations. *J. A. M. A.*, 89: 1735, 1927.
13. Carlson, H. A. Inhibition of respiration as a factor in the pathogenesis of postoperative pulmonary complications. *J. Thoracic Surg.*, 2: 196, 1932.
14. Best, C., and Taylor, N. B. Physiological Basis of Medical Practice. 2nd Ed. Baltimore, 1939. Williams & Wilkins.
15. Jackson, C. Postoperative pulmonary complications. *Internat. Clin.*, 4: 157, 1932.
16. Negus, V. E. Bronchoscopy in diagnosis and treatment of postoperative lung complications. *Brit. J. Anesth.*, 10: 165; *Proc. Roy. Soc. Med.*, 26: 1127.

# THE IMMEDIATE RESTORATION OF ACTIVE EXTENSION OF THE KNEE FOLLOWING FLEXION DEFORMITIES

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THE most common serious deformity in patients with rheumatoid arthritis is fixed flexion of the knee. When the disease has progressed to the subacute or chronic stage and the acute joint pain has subsided the patient frequently discovers that he is unable to stand because of 30 to 60 degrees of flexion deformity of the knee. There is usually 50 to 80 degrees of active and passive motion. *X*-rays show that the articular surfaces of the tibia and femur would be satisfactory for weight bearing if the knee could be extended normally.

The flexion deformity can be overcome by open lengthening of the posterior capsule and the biceps femoris tendon.[1] Following this procedure the knee can be passively extended to the normal limit. It has been the experience of the author that, following the restoration of full passive extension, the patient usually is unable actively to extend the knee through the final 10 to 20 degrees.

Normal extension of the knee is of no value unless it is under active muscle control. The knee must be held in full extension during that portion of the stride in which the full weight is carried on the extremity. If the patient is unable to do this his gait will be awkward and he will eventually have a recurrence of the flexion deformity.

The lack of complete active extension of the knee is due to overstretching of the patellar tendon during the years in which the knee was held in a flexed position or it is due to adhesions somewhere in the extensor muscle system which will not permit the patella to move far enough proximally to pull the tibia into proper alignment with the femur. Usually both these causes are responsible. It is impossible to locate or free the adhesions without further damage and scarring to the gliding mechanism of the extensor motor apparatus. The transplantation of the tubercle of the tibia by the author's technique offers a solution to the problem and permits immediate full active extension of the knee.

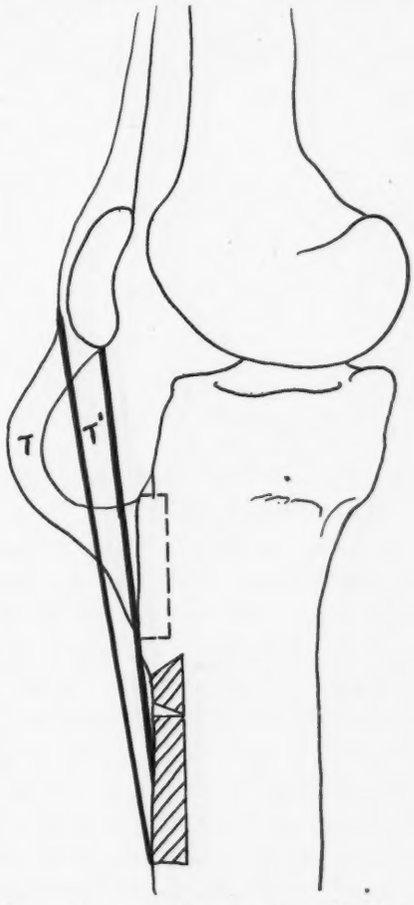


FIG. 1. T, patellar tendon showing laxity. $T^1$, patellar tendon after the transplantation of the tubercle of the tibia.

## TECHNIQUE

After the restoration of complete passive extension of the knee an incision is made

extending distally from the lower pole of the patella along the patellar tendon and the upper 3 inches of the anterior-medial surface of the tibia. The tendon (T, Fig. 1) is very lax, bulging forward a distance of ½ to ¾ inch. With the twin motor saw, parallel marks are made on the anterior-medial surface of the tibia extending distally from the tubercle for a distance of 2 inches. (3, Fig. 2A.) These marks are separated by about ½ inch and penetrate the cortex only far enough to scratch the surface. The distance between the twin saw blades is then increased ⅛ inch more and parallel marks are made on either side of the tubercle of the tibia extending from its distal border to the articular surface of the tibia. (2, Fig. 2A.) With the single motor saw, cuts are then made along these marks extending obliquely through the cortex so that the bone between the parallel cuts forms a dovetail (B1 and B2, Fig. 2A, B3 and B4, Fig. 2B). A transverse cut is then made across the dovetail just distal to the tubercle and another 2 inches below this across the distal end of the dovetail. With the twin motor saws separated by ¼ inch, a mark is made on the dovetail parallel with its lateral border extending from one transverse cut to the other. (1, Fig. 2A.) With the single motor saw this cut is continued obliquely through the cortex, medially, to form a narrow dovetail. (B5, Fig. 2A.)

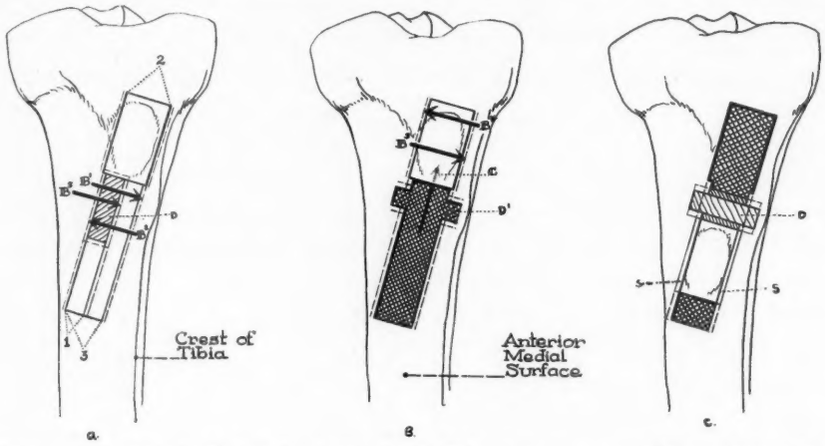


FIG. 2. Anterior-medial surface of the left tibia. A, 1, 2, 3, parallel marks made with twin motor saw. Parallel marks 2 are ⅛ inch farther apart than parallel marks 3. $B^1$, $B^2$, $B^5$, direction in which the oblique saw cuts are made with the single motor saw. D, dovetail graft ¼ inch wide. B, $B^3$, $B^4$, direction in which the oblique saw cuts are made with the single motor saw. C, direction of cuts made with the thin osteotome to separate the dovetail grafts from the cancellous bone beneath. $D^1$, gutter prepared to receive dovetail graft D. C, tubercle of tibia transplanted distally. The transverse dovetail graft D prevents the tubercle from slipping proximally. The shoulder S of the gutter prevents the anterior displacement of the tubercle.

With a thin osteotome the cortex between the parallel cuts is then freed from the cancellous bone beneath (C, Fig. 2B) and the portion between the transverse cuts removed. By the same technique a transverse gutter is prepared (D1, Fig. 2B) to receive the small dovetail graft. (D, Fig. 2A.) The tubercle is then displaced distally a distance of ⅛ inch for each 10 degrees of fixed flexion which was present before the posterior capsulotomy operation and the ¼ inch wide dovetail (D, Fig. 2C) placed transversely across the gutter proximal to the tubercle.

The double dovetail technique holds the tubercle solidly in its new position and prevents its displacement anteriorly and proximally.

This method of transplantation of the tubercle of the tibia requires a very accurate cabinetmaker's fit between the dovetail grafts and the gutters into which

they are inlaid. The parallel marks made on either side of the tubercle are ⅛ inch farther apart than those for the gutter into which the tubercle is to be inlaid to allow for the sawdust lost in cutting the dovetails. This insures a tight fit. As soon as the transverse dovetail is in place full active and passive flexion and extension of the knee can be started without fear of avulsion of the transplanted tubercle.

## SUMMARY

1. After the restoration of normal passive extension in fixed flexion deformities of the knee the patient is frequently unable actively to extend the knee through the final 10 to 20 degrees.

2. Transplantation of the tubercle of the tibia distally ⅛ inch for each 10 degrees of fixed flexion deformity which was present before the posterior capsulotomy operation restores full active extension of the knee.

3. A new technique for transplantation of the tubercle is presented. By this method the tubercle is held in place by double dovetails.

4. This technique permits immediate active extension of the knee to the normal limits and eliminates the necessity of waiting three or four weeks for callus to unite the transplanted tubercle to the tibia.

## REFERENCE

1. Wilson, P. D. Posterior capsulotomy in certain flexion contractures of the knee. *J. Bone & Joint Surg.*, 11: 40, 1929.

# UNEXPECTED OCCURRENCE OF SUGAR IN THE URINE FOLLOWING GLUCOSE INFUSIONS*

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IN a far larger percentage of cases than is ordinarily realized, sugar can be demonstrated in the urine of patients soon after they have been given intravenous infusions of glucose. This is so even though the usually accepted tolerances for sugar have not been overstepped at any time during the infusion and, indeed, occasionally though the maximum tolerated dose of sugar has not even been approached. This phenomenon, which has apparently plagued careful observers[1] seems explicable on the basis of available experimental observations, though this statement is by no means meant to imply that the entire story of glucose metabolism is yet known.

*Utilization of Sugar.* Under conditions of glucose administration short of the production of glycosuria all of the administered glucose is considered to be "utilized." Maximal administration of glucose in the absence of glycosuria produces a condition of maximal utilization.

When maximal utilization is occurring there is acceptable evidence, based on the measurement of heat production, that from one-quarter to one-third of the administered glucose is immediately oxidized to carbon dioxide and water, whereas the remaining two-thirds to three-quarters, although it ultimately undergoes oxidation, is first stored in the body in one form or another for a variable period of time.[2] Most of the storage is generally believed to occur in the form of glycogen, but conversion to other compounds is not only chemically possible but physiologically practically certain—conversion to fat, to lactic acid, and to other substances such as hexosephosphate, glycerophosphate and glucosamine.

Thus "utilization" is a term which is equivalent to "oxidation" in the long run, but as far as shorter periods of time are concerned includes both "oxidation" and "storage."

*Glucose Tolerance.* When glucose is administered in larger quantities, i.e., at faster rates, than it can be utilized by the body the excess is excreted in the urine. Under such conditions utilization can be computed by subtracting the quantity of sugar appearing in the urine from the total quantity of sugar administered during any particular period of time. On the assumption that glucose added directly to the blood stream by artificial means is treated by the body in no essentially different manner from that which is absorbed in the natural way from the intestine, and by using an accurately metered pump, constant rates of injection, and the intravenous infusion technique, it is possible to determine accurately the rate of glucose administration which produces maximal utilization. Thus has been established the widely quoted and generally accepted dictum that glucose can be completely utilized if the rate of administration does not exceed about 0.85 Gm. per Kg. of body weight per hour[3] and that techniques which do not exceed such a rate of administration can be used in full confidence that the critical level of sugar in the blood will not be exceeded and therefore sugar will not appear in the urine.

Obviously, direct addition of glucose to the blood stream, as in intravenous infusion, constitutes a highly artificial situation against which the body's defenses

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have not been developed by ordinary evolutionary processes; thus a patient's glucose tolerance is quite at the mercy of the infusion technique. Whether the absorption rate from the intestine is great enough to produce "alimentary glycosuria" in normal individuals is a disputed point. Apparently the absorption rate in some species may be as high as 1.6–1.7 Gm. per Kg. body weight per hour, but even at this high rate of absorption glycosuria does not occur.[4] At present the consensus of opinion seems to regard the occurrence of "alimentary glycosuria" as always an evidence of impaired tolerance.

The magnitude of the rise in blood sugar level after measured, oral or intravenous administration of glucose, the time relationships of the initial rise and subsequent fall in the blood sugar level, the amount of sugar which appears in the urine, and the proportions of the retained sugar which are on the one hand oxidized and on the other hand stored, all conform to certain general standards which form the basis for the familiar "glucose tolerance tests."

This much of the story of glucose metabolism is quite familiar to all students of the subject, but it does not seem to be widely appreciated that the above-mentioned concepts hold good only for a body in a relatively normal functional state. The particular considerations which do not seem to have been sufficiently emphasized are: (1) that abnormal reactions may be expected under conditions of gross imbalance between the secretions of the endocrine glands controlling carbohydrate metabolism, viz., pancreas, pituitary gland, adrenal glands; (2) that glucose utilization and glucose tolerance vary somewhat widely with the stage of nutrition; and (3) that glycosuria or lack of glycosuria is in part dependent upon the function of the kidneys and particularly upon the magnitude of diuresis.

Unfortunately it does not seem possible at the present time, in spite of a multitude of experimental investigations on the endocrine control of carbohydrate metabolism, to discuss profitably the part which may be played by unrecognized endocrine imbalance in the production of glycosuria after glucose infusions. This is true not only because the interrelationship of the various endocrine secretions is still much in dispute but also because no sufficiently simple and practical means has yet been devised for rapid routine assay of the endocrine balance of the individual patient. Fortunately, at least as far as this discussion goes, however, it does not seem necessary to invoke any such complicated hypothesis to explain most cases of post-infusion glycosuria. The known effect of the pre-infusion level of pancreatic activity on the utilization of infused glucose alone seems adequate to account for the major portion of the cases.

*The Effect of Nutrition on Sugar Utilization and Glucose Tolerance.* That undernutrition and starvation depress glucose utilization has been appreciated in a general way since 1873, the year in which Lehmann demonstrated that rabbits, previously fasted for two or three days, exhibited glycosuria following intravenous injection of sugar in amounts which caused no glycosuria in animals which had been regularly fed.[2] Hofmeister in 1890 confirmed the observation that fasting or under-nourished animals show impaired glucose assimilation and gave to the condition the name "hunger diabetes."[2]

With this observation as a point of departure and after much experimental observation the thesis has been gradually developed that sugar utilization is very definitely a biologic variable. When the body is in nutritional equilibrium the utilization of carbohydrates by the tissues shows a relatively exact automatic adjustment to the supply of sugar. Under these conditions not only is there both an adequate supply of glucose and an adequate utilization thereof, but periods of moderate increase of supply are followed by corresponding periods of increased utilization. If, on the other hand, the body has been conditioned by a moderate period of

fasting to diminished sugar utilization, and sugar is now administered in moderate amounts the decreased tolerance established by the interval of fasting becomes manifest in the development of a hyperglycemia which not only attains its maximum more rapidly than normal but attains a higher value as well. Furthermore, if the period of diminished carbohydrate supply or actual fasting exceeds a certain critical value, the hyperglycemia which follows subsequent administration of glucose will be great enough to exceed the kidney threshold for glucose and one or another degree of glycosuria will result. In this connection it should be emphasized that the loss of sugar which occurs is in spite of the need of the body for the glucose thus being wasted.

If one continues to supply glucose beyond this point indefinitely, i.e., if the period of fasting is brought to a close, glucose utilization may be expected, other things being equal, to increase progressively and ultimately regain its normal level. The glycosuria is transient and disappears as soon as the rate of utilization increases to a point where the blood sugar level is kept within suitable bounds. In short, the body returns to its normal carbohydrate equilibrium. Peculiarly enough, however, if the opposite course is pursued, if carbohydrate administration is stopped, and starvation is allowed to progress, the response to short periods of glucose administration under such conditions shows no further lessening of carbohydrate utilization, but on the contrary an enhancement. The explanation undoubtedly is that as food curtailment passes over into actual starvation carbohydrate tolerance actually improves because a new source of carbohydrate is made available by virtue of conversion of the body's own protein to sugar. As long, therefore, as endogenous protein is being converted to carbohydrate the ability of the body to utilize sugar is to some extent anomalously regained. Terminally, of course, carbohydrate utilization permanently diminishes as the supply of available protein becomes exhausted, but this is not particularly pertinent here.

The point which is of the utmost significance in the present discussion is that complete deprivation of carbohydrate food for short periods of time, or curtailment of the supply of such food over a longer period of time profoundly alters the utilization of glucose and predisposes the body to the elimination of sugar in the urine when carbohydrate administration is resumed even though the amount of carbohydrate is considerably less than that required to produce glycosuria normally. In short, the moderately sick person whose food supply has been curtailed or completely cut off for a short time because of alimentary derangements and who is likely therefore to be given glucose parenterally is precisely the person most likely to show "unexplained" glycosuria. The person who has undergone more prolonged glucose curtailment is not so likely to show glycosuria.

*The Effect of Diuresis on Glucose Utilization and Glycosuria.* Inasmuch as the parenteral administration of glucose inevitably involves concomitant administration of water, the glucose necessarily being given in the form of a solution, the question naturally arises whether the degree of dilution of the infused glucose makes any difference in its utilization. If the answer to this question could be given with assurance it would throw light on the possibly important practical decision whether to administer glucose in any given case in concentrated form or in more generous dilution.

This matter has received but scant attention, partly no doubt because of the technical difficulties involved in devising crucial experiments. Not only does a period of preliminary curtailment of glucose diminish the utilization of the substance when it is subsequently administered, as has been previously described, but preliminary administration of glucose even for a short period of time increases the

utilization of this substance as its administration is continued. This phenomenon has been called "priming." The most obvious explanation is that the islet cells of the pancreas are capable of elaborating the substance which controls the level of blood sugar, insulin, at various levels of activity. Thus, if these cells happen to be functioning at some particular relatively low level and glucose is suddenly supplied in quantity the level of insulin elaboration may be temporarily insufficient to produce complete utilization and glycosuria occurs. If, however, glucose continues to be supplied in not too great quantity insulin elaboration is stimulated, utilization becomes stabilized at a higher level and glycosuria disappears.

Until recently there has been no good evidence that the dilution of administered glucose significantly affects utilization. Only one animal from the experiments of the earlier workers with the constant or metered-pump method of administering glucose solutions showed results capable of interpretation from this point of view and the protocol from this animal seemed to indicate that the dilution of the glucose did not affect either utilization or excretion, but on the other hand, the degree of diuresis was not great.[5] A later worker was able to conclude from a series of experiments in which 5 per cent to 80 per cent solutions of glucose were used as infusion vehicles that the administration of much water along with the glucose, though it promotes diuresis, increases retention of glucose in the body and thus tends to prevent glycosuria.[6] At a given level of blood sugar, however, it was concluded by this worker that when more water is available more glucose is excreted in the urine.

When conditions are carefully controlled, it lately seems to have been shown experimentally that great diuresis definitely decreases glucose utilization, i.e., both decreases the quantity of glucose retained in the body and increases the quantity of glucose lost in the urine.[7] In order to make the findings conclusive it was necessary to stabilize the activity of the islet cells of the pancreas at a high level by initial priming with glucose solution at a constant rate of injection and then, without changing the rate of glucose injection, to add large quantities of saline solution also at a constant rate.

Glycosuria, due to diuresis, probably does not represent any alteration of carbohydrate metabolism but, instead, is dependent upon increased filtration through any given glomerulus and perhaps also the opening of previously inactive glomeruli. There is evidence that the kidney tubules are able to reabsorb from the tubular urine only a certain maximal quantity of glucose per unit interval of time. If the glomeruli pass a filtrate so rich in glucose that tubular reabsorption is inadequate to restore it all to the blood stream or if the quantity of filtrate is so great that its sojourn in the tubules is too short to permit complete reabsorption, glycosuria results.

At all events, if the experimental results can be accepted it seems that the amount of glucose excreted in the urine during an infusion may be increased as much as ten times by diluting the infusate without changing the rate of administration of the glucose as such.

## CONCLUSIONS

1. In general the presence of glucose in the urine of a patient is an indication that there is more sugar in the blood stream of that patient than the body can immediately oxidize or store, i.e., more than the body can "utilize."

2. In nondiabetic subjects and under ordinary or normal conditions of nutrition utilization is at a fairly uniform level from person to person. The limits of utilization are probably never exceeded in such circumstances if glucose is added to the blood stream whether from the gastrointestinal tract or parenterally in amounts not exceeding 0.75 Gm. per Kg. body weight per hour.

3. Ability of the body to utilize glucose, however, is markedly affected by the amount of glucose which has been available previously; thus a period of glucose curtailment or denial (starvation) decreases the ability of the body to utilize this substance subsequently, and conversely the administration of moderate amounts of glucose during a preliminary period of time enhances the utilization of this substance subsequently.

4. Most patients who receive glucose parenterally are given this substance precisely because they have shown themselves incapable of taking, through natural channels, carbohydrate food over a period of time preceding the decision to make up the deficit artificially. Such patients are more than likely to exhibit diminished capacity for glucose utilization, and if they are treated as if they can utilize the substance as fast as normal individuals, they may be expected to develop glycosuria.

5. Inasmuch as the mere administration of glucose over a period of time, probably by stimulating the internal secretion from the cells of the islands of Langerhans, increases the level of glucose utilization, a patient who shows initial slight glycosuria during suitable parenteral administration of a solution of glucose may cease to show sugar in the urine after his utilization level has been restored to normal by the judicious continuation of the administration.

6. Inasmuch as excessive diuresis may cause the appearance of sugar in the urine in cases in which glycosuria would not otherwise appear, and may increase the elimination of sugar in cases in which the elimination of sugar would otherwise be less, glycosuria is more likely to occur and occur more extensively in those cases in which glucose is administered in dilute solution or in conjunction with large quantities of saline solution.

7. There is good experimental precedent, therefore, for individualizing cases in the parenteral administration of glucose solutions in order to avoid glycosuria. When patients are in a state of partial glucose starvation, as most sick patients are, it is suggested that they be regarded as having an abnormally decreased ability to utilize this substance and accordingly that they be given only moderate amounts of 5 per cent solutions, or perhaps, better still, 10 per cent solutions for several hours in an attempt to build up their level of utilization, before resorting to the administration of more dilute solutions of glucose or quantities of saline solution to combat associated dehydration. The rate of administration of glucose might well be kept for some hours at not much more than half the maximal accepted rate for persons in a normal state of nutrition, i.e., 3, 4 or 5 Gm. per Kg. body weight per hour. On the other hand, if the combating of dehydration rather than the administration of glucose for nutritional purposes is the pressing indication, it might be preferable to confine one's parenteral administration initially to saline solutions for several hours, thereafter building up a suitable tolerance for glucose gradually by interspersing periods of relatively slow administration of 5 to 10 per cent solutions of glucose alone.

## REFERENCES

1. Fantus, B. Fluid postoperatively; statistical study. *J. A. M. A.*, 107: 14, 1936.
2. Chambers, W. H. Undernutrition and carbohydrate metabolism. *Phys. Rev.*, 18: 248, 1938.
3. Woodyatt, R. T., Sansum, W. D. and Wilder, R. M. Prolonged and accurately timed intravenous injections of sugar. *J. A. M. A.*, 65: 2067, 1915.
4. Cori, C. T. Fate of sugar in the animal body. I. The rate of absorption of hexoses and pentoses from the intestinal tract. *J. Biol. Chem.*, 66: 691, 1925.
5. Sansum, W. D. and Woodyatt, R. T. Studies on the theory of diabetes, VI.I. Timed intravenous injections of glucose at lower rates. *J. Biol. Chem.*, 30: 155, 1917.
6. Davis, D. M. Effect of dextrose given intravenously on blood composition and urinary secretion. *Am. J. Physiol.*, 43: 514, 1927.
7. Cutting, R. A., Larson, P. S. and Lands, A. M. Effect of diuresis on the excretion of glucose in the normal dog during constant glucose infusion. *Am. J. Physiol.*, 124: 662, 1938.

# CIRCULATION TIME IN SURGICAL PATIENTS*

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EVER since Sir William Harvey's[1] epochmaking description of the circulation of the blood in 1628, the matter of blood velocity, or its inverse ratio, circulation time, has been investigated by a number of workers. We have studied circulation time by the sodium cyanide method in a large number of surgical patients. Our endeavor has been to find what value, if any, this test has from a prognostic standpoint in surgical morbidity or mortality.

Blumgart,[2] in his extensive monograph, has given an excellent historical survey covering in detail all investigation up to the time of his publication. A brief résumé of the work on circulation time to date is here presented.

Hales,[3] in 1733, computed the velocity of blood flow from his estimation of the capacity of the left ventricle, the diameter of the base of the aorta and the pulse rate. The same investigator made microscopic observations on the capillaries of the lungs in experimental animals and estimated blood velocity there by this means.

Hering,[4] in 1827, injected postassium ferrocyanide into the jugular vein of a horse and measured circulation time by noting the time of the appearance of the ferrocyanide in the opposite jugular. His average jugular to jugular circulation time in horses was 26.2 seconds.

Volkman,[5] in 1850, described the use of the hemodromometer which gauged blood velocity by recording the movement of a pendulum placed in the lumen of a blood vessel.

Vierordt,[6] in 1858, designed his hemotachometer. He modified Hering's method and worked on rabbits, cats, dogs and other experimental animals. It was his impression that circulation time is related to pulse rate, and from such observations in animals estimated the jugular to jugular circulation time in man to be 23.1 seconds if the heart rate were 72.

Stewart,[7] in 1893, injected hypertonic saline intravenously into one jugular vein and measured circulation time by noting electrical conductivity changes in the blood in a vascular bed at a distance from the site of injection. He also injected methylene blue into one vein and timed its appearance by transillumination in the opposite carotid artery.

Bornstein,[8] in 1912, had patients breathe 7 per cent carbon dioxide and measured the time for the patient's respirations to quicken. He estimated that this was half the circulation time. This deduction was based on the fact that the time as measured was the time necessary for the carbon dioxide to travel from the pulmonary bed to the respiratory center or approximately half the total circulation.

Loevenhart, Schlomowitz and Seybold,[9] in 1922, measured the circulation time in rabbits and dogs with sodium cyanide. The same workers reported on the use of lithium salts, hexamethylene and ferrocyanide. Koch,[10] in 1922, injected fluorescine and noted the time of arrival in the opposite forearm by drawing repeated blood samples. Hirschsohn and Mandel,[11] in 1922, injected calcium chloride and took as their end point a sense of warmth or burning in the throat.

Blumgart and Yens,[12] in 1927, described their method of injecting a radioactive substance and noting its appearance in various parts of the cardiovascular bed by radiographic means. This method, though highly technical, allowed for measurements in

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parts of the circulation. Weiss, Robb and Blumgart,[13] in 1929, injected histamine and noticed its peripheral vasodilator effect to measure circulation time.

Klein and Heineman,[14] in 1929, reported on a method similar to that of Koch[10] but used congo red instead of fluorescine. Winternitz, Deutsch and Brull,[15] in 1931, injected sodium dehydrocholate and took as their end point the appearance of a bitter taste. Tarr, Oppenheimer and Sager,[16] in 1933, reported on the use of the same material in a variety of medical conditions. Robb and Weiss,[17] in 1933, reported on the use of sodium cyanide to measure circulation time in man. This work will be mentioned in detail below.

Fishberg, Hitzig and King,[18] in 1933, reported on the injection of saccharin and timed their patients by the appearance of a sweet taste. Hitzig,[19] in 1934, reported on the injection of ether. By noticing the odor of ether on the patient's breath, he measured the circulation time for the lesser or pulmonary circulation. Goldberg,[20] in 1936, injected calcium gluconate and timed the circulation in various regions by having the patient report on the appearance of a burning sensation in the pharynx, face, chest, perineum, hands and feet.

An ideal method of measuring circulation time on a busy surgical service falls within rather narrow bounds. It should be inexpensive. It should be easily performed by the average house officer on a surgical service. It should not entail the use of instruments or devices uncommon to a surgical ward. Injection of a chemical into the antecubital veins falls well into line. The material should be nontoxic. It should not, of itself, influence the rate of blood flow. The reaction should produce objective and not subjective signs. The reaction time compared to the circulation time should be minimal, or better, negligible. (It is to be noted that the circulation time, as measured, is the time in seconds from the time of injection to the appearance of the objective reaction. This includes the time consumed in producing the objective reaction after the material has arrived at a given point.) The reaction should be short-lived to allow for frequent repetition. It must not produce any element of fear in the patient. The reaction should occur without relation to the presence or absence of morbid states in the body.

Sodium cyanide, in proper dosage, has been found to fit very closely the requirements here presented. As early as 1828 Ryan[21] described the use of sodium cyanide as a respiratory stimulant. Loevenhart, Lorenz, Martin and Malone,[22] in 1918, reported on the stimulation of respiration by sodium cyanide. They found the dose necessary to produce respiratory stimulation to be, in an adult, about 3 to 5 mg. which was equivalent to about 0.04 to 0.07 mg. per Kg. of body weight. Experimenting with dogs, they found the fatal dose of sodium cyanide to be about twenty times the therapeutic dose. They estimated that this relation probably holds true for man. They state that sodium cyanide is destroyed in man at the rate of 1.5 to 2.0 mg. per minute. It is converted into innocuous sulfur compounds and nitrogen compounds. It is excreted in the breath as hydrocyanic acid and in the urine as cyanide and sulfocyanide.[23] Robb and Weiss, mentioned above, studied the use of sodium cyanide to determine circulation time in man. They found that doses of 0.11 mg. per Kg. of body weight were safe and effective. Such dosage produced a definite objective end point—a mild increase in respiratory rate and a marked increase in amplitude of respiration. Toxic doses in animals will produce first a fleeting stimulation of respiration which is followed quickly by a rapid, shallow and irregular respiration followed by respiratory paralysis and death.

Heymans, Bouchaert, and Dautreband[24] and Owen and Gesell[25] state that the action of sodium cyanide in stimulating respiration is on the carotid sinus and not on the respiratory center. Their evidence in support of this finding is a series of denervation experiments on animals. Tschibukmacher[26] in an elaborate anatomic dissection of the

carotid sinus nerve and its connections describes pathways between the carotid sinus nerve and the vagus. It may be over such a pathway that impulses travel to produce the respiratory response. Robb and Weiss support the view that action is on the carotid sinus, also by indirect evidence. They measured, simultaneously, cyanide time and the time required for glucose to reach the femoral artery when both are injected in one solution. For the details on this work we refer the reader to the original article.

We conducted about 800 circulation time tests on about 500 surgical patients of all types whose ages ranged from 10 to 85 years. We varied our dosage with weight in the first fifty cases, but found little indication for so doing and decided on three definite doses to be used throughout: 8 mg. for males, 6 mg. for females and 4 mg. for children. We used a 2 per cent solution and thus were injecting 0.4 c.c., 0.3 c.c. and 0.2 c.c. in men, women and children, respectively. We injected routinely into the right antecubital vein and used a tuberculin syringe. With very few exceptions, manual compression was used instead of a tourniquet. The patient was always lying flat in bed, head on one pillow, with the right arm at the level of the auricles. We found that time of day did not affect results, nor did the proximity to meals have any effect. Our injection time was always under one second.

In almost 100 cases we measured blood pressure before and after taking circulation time measurements. We found no substantial change at any time, before or after. There was some increase in pulse rate, up to about ten beats per minute (except in cases of hyperthyroidism where the increase averaged eighteen beats per minute) for a short while after the test. This in most instances in our experience was concomitant with an expression of anxiety and may have been on a psychogenic basis. Some of our normal subjects were senior medical students who watched the investigation in ward patients and understood the procedure. In these cases there was no appreciable change in pulse rate. We experienced no untoward reactions in the entire series. When it was desired to repeat the test, in no case was the patient unwilling to undergo repetition.

To gain some degree of facility in recognizing the end point early in this series, values were accepted only, when on repetition on the test, the results checked within two seconds. In 130 healthy, adult subjects* ranging in age from 20 to 40 years, our average circulation time was 17.0 seconds, with readings ranging from 10 to 26 seconds. Robb and Weiss report as their average in healthy normals 15.6 seconds with values ranging from 9 to 21 seconds. Weiss, Robb and Blumgart, using 0.001 mg. of histamine phosphate per Kg. of body weight (in 1:5000 solution) and using a flushing of the face as their end point, report sixty-five normals averaging 23 seconds with readings ranging from 13 to 30 seconds. Their explanation for the discrepancy (6.5 seconds in recorded experiments) is that the cyanide time measures time from the periphery to a large vessel (the carotid artery) whereas histamine measures the time to a more peripheral site and includes passage through the capillary bed to the venules, where the histamine acts. However, Tarr, Oppenheimer and Sager using sodium dehydrocholate averaged 13 seconds and Goldberg using calcium gluconate averaged 12.5 seconds for normals. Their points of action were the peripheral neural end organs in the pharynx.

In reviewing our figures, we noted some correlation between circulation time by the cyanide method and the subjects' age. Table 1 is an analysis on an age basis, including all subjects examined. The only prognostic significance here presented is the scientific evidence of a generally increasing circulation time with advancing years. It simply supports the view that early general

* In the "normal" group were medical students and patients not seriously ill, admitted with such conditions as contusions and abrasions, sprained ankles, simple inguinal hernia and the like. Robb and Weiss included similar patients in their normals.

motion in postoperative patients of advanced years is advisable if we are to avoid the dangers consequent upon stasis.

TABLE I
RELATION OF AGE TO CIRCULATION TIME

| Age Group, Years | No. of Cases | Average, Seconds | Range, Seconds |
|---|---|---|---|
| 10 to 19 | 80 | 13.0 | 8 to 22 |
| 20 to 29 | 105 | 16.0 | 10 to 22 |
| 30 to 39 | 105 | 17.1 | 10 to 22 |
| 40 to 49 | 100 | 18.9 | 10 to 27 |
| 50 to 59 | 190 | 21.9 | 12 to 34 |
| 60 to 69 | 110 | 25.0 | 13 to 37 |
| 70 to 79 | 50 | 23.0 | 14 to 34 |
| 80 to 85 | 20 | 27.0 | 18 to 35 |

It is of interest to quote here from Harvey's[1] work: "The circuit of the blood is accomplished now more rapidly, now more slowly according to temperment, age, . . . of the individual . . . "

Analysis of our records on the basis of general type of disorder including all age groups is given in Table II. Inflammatory

TABLE II

| Type of Disorder | No. Cases | Average, Seconds | Range, Seconds |
|---|---|---|---|
| Inflammatory conditions... | 320 | 17.8 | 10 to 37 |
| Traumatic conditions..... | 290 | 19.0 | 8 to 35 |
| Neoplastic conditions..... | 80 | 21.3 | 10 to 36 |
| Peripheral vascular disease. | 60 | 22.0 | 17 to 28 |
| Hyperthyroidism..... | 15 | 8.9 | 6 to 12 |
| Patients with various surgical conditions whose E.-K.G. read—were normal. | 30 | 25.2 | 13 to 40 |

conditions included such an expanse as suppurative adenitis in children of 10 to carbuncles in diabetics of 70. Traumatic conditions included fractured hips in many elderly patients and various types of fractures in children. Neoplastic conditions were, with the exception of one male, age 23, with inoperable carcinoma of the rectum, all in the age groups above 40. Peripheral vascular disease included such conditions as Buerger's disease, Raynaud's disease, diabetic arteriosclerosis, senile arteriosclerosis and traumatic gangrene of the foot following fracture of the leg. These patients were for the most part past the 40-year age group. The abnormal electrocardiogram readings included such interpretations as: myocardial fibrosis, myocardial damage, sinus tachycardia, auricular fibrillation and healed infarct from coronary occlusion. These were also in the older age groups.

During the time of this investigation there were two wound disruptions, (both in peptic ulcers which had been ruptured six hours prior to operation) and ten wound infections. Investigation of the circulation time in these cases revealed that circulation time adhered to the average for the age group within 10 per cent. In this same period there were seven patients with pulmonary complications following general anesthesia. Their circulation times were normal both before operation and after the subsidence of their pulmonary complication. In this regard Brandt[27] reports 153 patients with pulmonary tuberculosis with an average circulation time by the saccharin method of 12.73 seconds. Patients who had particularly stormy postoperative courses were compared with patients whose postoperative courses were uneventful. There was no essential difference in circulation time. Review of the circulation time of those patients who died as the result of their operations or complications indicated no unusual findings.

Patients in cardiac decompensation or with cardiac histories and impending decompensation were not admitted to this service. We offer no data on such patients.

## DISCUSSION

Our values for circulation time seem to be a bit higher than those of other workers using this method and also higher than results obtained by most other injection methods. Our study was aimed at determining the prognostic value of this test. The same end point was used in all cases. Hence our values are of significance regardless of the base line for normal.

## SUMMARY AND CONCLUSIONS

1. Many methods for measuring circulation time are reviewed.

2. The injection of a substance into a peripheral vein for determining circulation time appears to be an ideal method for a large series of tests.

3. The attributes of an ideal substance for injection are stated.

4. Sodium cyanide in proper dosage conforms to this ideal.

5. The pharmacology of sodium cyanide as it relates to its use in circulation time measurement is presented.

6. The test has been applied to about 500 patients about 800 times.

7. Our average values are slightly higher than those of other observers.

8. There is a correlation between age and circulation time, according to our figures, that is, the circulation time increases with age. The average rate in the age group for 80 to 85 years is twice that for the age group 10 to 20 years.

9. This test does not afford any prognostic significance in surgical patients either from the morbidity or mortality standpoint.

## REFERENCES

1. Harvey, W. Exercitatio anatomica de motu cordis et sanguinis in animalibus. English Translation by Chauncey D. Leake. Springfield, Ill. 1931. C. Thomas.
2. Blumgart, H. L. Velocity of blood flow in health and disease. *Medicine*, 10: 1, 1931.
3. Hales, S. Statical Essay, Vol. 2. London, 1733. Quoted by Blumgart.[2]
4. Hering, E. *Ztschr. f. Physiol.*, 3: 85, 1827.
5. Volkman, A. W. Die Hemodynamik nach Versuchen, p. 185. Leipsig, 1850. Quoted by Blumgart.[2]
6. Vierordt, K. Die Erscheinungen und Gesetze der Stromgeschwindigkeiten des Blutes. Frankfurt A.M., 1858. Quoted by Blumgart.[2]
7. Stewart, G. N. Researches on the circulation time in organs and on the influences which affect it. 1. Preliminary paper. *J. Physiol.*, 15: 1, 1894.
8. Bornstein, A. Ueber die Messung des Krieslaufzeit in der Klinik. *Behandl. d. Kong. f. inn. Med.*, 29: 457, 1912.
9. Loevenhart, A. S., Schlomowitz, B. H. and Seybold, E. G. Determination of circulation time in rabbits and dogs and its relation to reaction time of respirations to sodium cyanide. *J. Pharmacol. & Exper. Therap.*, 19: 221, 1922.
10. Koch, E. Die Stromgeschwindigkeit des Blutes. *Deutsch. Arch. f. klin. Med.*, 140: 39, 1922.
11. Hirschsohn, J., and Mandel, H. Notiz zur Kenntnis der Hämmodynamik beim Pneumothorax. *Beitr. c. Klin. d. Tuberk.*, 49: 64, 1922.
12. Blumgart, H. L., and Yens, O. C. Studies on velocity of blood flow. 1. Method Utilized. *J. Clin. Investigation*, 4: 1, 1927.
13. Weiss, S., Robb, G. P. and Blumgart, H. L. Velocity of blood flow in health and disease as measured by effect of histamine on minute vessels. *Am. Heart J.*, 4: 1, 1929.
14. Klein, O. and Heineman, J. Zur Messung due Stromschwindigkeit des Blutes beim Menschen. *Zentralbl. f. Inn. Med.*, 50: 490, 1929.
15. Winternitz, M., Deutsch, J. and Brüll, Z. Eine klinische Brauchbare Bestimmungsmethode der Blutumlaufzeit mittals Decholihinjektion. *Med. Klin.*, 27: 986, 1931.
16. Tarr, L., Oppenheimer, B. S. and Sager, R. O. Circulation time in various clinical conditions determined by use of sodium dehydrocholate. *Am. Heart J.*, 8: 766, 1933.
17. Robb, G. P. and Weiss, S. Method for measurement of velocity of pulmonary circulation and peripheral venous blood flow in man. *Am. Heart J.*, 8: 650, 1933.
18. Fishberg, A. M., Hitzig, W. M. and Kings, F. H. Measurement of circulatime with saccharin. *Proc. Soc. Exper. Biol. & Med.*, 30: 651, 1933.
19. Hitzig, W. M. Measurement of circulation time from antecubital veins to pulmonary capillaries. *Proc. Soc. Exper. Biol. & Med.*, 31: 935, 1934.
20. Goldberg, S. J. Use of calcium gluconate as a circulation time test. *Am. J. M. Sc.*, 192: 36, 1936.
21. Ryan. *Tr. Coll. Physicians, London*, 1828.
22. Loevenhart, A. S., Lorenz, W. F., Martin, H. G. and Malone, J. J. Stimulation of respiration by sodium cyanide and its clinical application. *Arch. Int. Med.*, 21: 109, 1918.
23. Cohen, S. S., and Githens, T. S. Pharmacotherapeutics, Materia Medica and Drug Action. New York, 1928.
24. Hyman, E., Bouchaert, J. J. and Dautrebrande, L. Sinus carotidien et reflexes respiratoires. III. Sensibilité des sinus carotidiens aux substances chimique. Action stimulante respiratoire reflexe du cyanure de sodium, du cyanure de potassium, de la nicotine et de la loudline. *Arch. internat. de pharmacodyn. et de therap.*, 40: 54, 1931.
25. Owen, H. and Gesell, R. Peripheral and central chemical control of pulmonary ventilation. *Proc. Soc. Exper. Biol. & Med.*, 28: 756. 1931.
26. Tschibukmacher, N. B. Surgical anatomy of the carotid sinus nerve and intercarotid ganglion. *Surg., Gynec. & Obst.*, 65: 740, 1938.
27. Brandt, M. A., and Hurst, A. A study of venous pressure and circulation time in pulmonary tuberculosis. *J. Thoracic Surg.*, 6: 638, 1937.

# PRACTICAL APPLICATION OF THE FALLING DROP METHOD FOR DETERMINING SURGICAL PROGNOSIS*

## PRELIMINARY REPORT

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THE proper administration of fluids in cases of dehydration, edema and shock has been and is a vexing problem. Various methods for the maintenance of proper fluid balance are at present employed, but most of these are inadequate, and dehydration or edema may still suddenly develop in any case under treatment.

We have devised a method for determining the status of the fluid balance which can be carried out in a few minutes by a trained intern or laboratory staff member. Tissue and pulmonary edema and massive internal hemorrhage can be predicted twenty-four to forty-eight hours before they appear clinically. When plasma protein (which follows accurately the specific gravity of the plasma as well as that of whole blood) approaches the so called "critical level" of 5.5 Gm. per cent, oncoming edema should be suspected. Edema will appear if the reading goes below that level, although it may be several days after this level is reached before edema is clinically apparent. When the critical level is reached, however, fluids should be stopped and a small blood transfusion given. This prevents the development of edema and returns the specific gravity of the blood, the plasma, and the blood protein to their respective normals.

It is now generally accepted that the specific gravity of whole blood and blood plasma varies with the degree of dehydration, edema, shock, or hemorrhage. The first attempt to measure the specific gravity of whole blood was made in the seventeenth century by Robert Boyle. Since then numerous other methods have been devised. In 1924, Barbour and Hamilton[1] described a simple method of measuring the specific gravity of whole blood, blood plasma, and other body fluids. This method is considered by many to be the most practical to date. It is based upon the principle that the time required for a drop of a certain volume of fluid to fall a fixed distance through a liquid (bromobenzenexylene solution) depends upon the density of the drop and that of the fluid through which it is falling. This is compared with a determination which is made with a standard solution of potassium sulfate of known density, the specific gravity of which closely approximates that of normal whole blood. The density of the bromobenzenexylene solution is adjusted so that the falling time of the drop of blood is from 20 to 60 seconds. The drop of blood must be 0.01 ml. by volume and the distance of the fall 30 cm.

The normal average value of the specific gravity of whole blood as determined by this method is 1.0566 for males and 1.0533 for females. The maximum concentration usually occurs in the morning, varying about 0.0033 during the course of the day.

The specific gravity of blood plasma is determined by the same method. Plasma is best obtained by adding approximately 5 mg. of heparin to 5 c.c. of venous blood. The heparinized blood is centrifuged and the supernatant fluid is used for the determination. The specific gravity of normal plasma, as determined by this method, is 1.0270 which corresponds to a plasma protein of 6.83 Gm. per cent.

Moore and Van Slyke[3] showed the constant relationship between the specific gravity of blood plasma and the plasma protein

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content in the edema of nephritis. This was essentially confirmed by the studies of Weech, Reeves, and Goettsch.[4] They established a formula from which the values of plasma protein could be derived from the determination of the specific gravity of the plasma: (plasma specific gravity 1.0069) × 340.1 = plasma protein.

The present investigation was undertaken to determine the practical application of the Barbour and Hamilton method for the determination of the specific gravity of whole blood and blood plasma as a clinical aid in diagnosis, prognosis, and treatment of patients at the Cumberland Hospital who presented evidence of shock, edema, dehydration or hemorrhage.

Table I
NORMAL VALUES

| | | |
|---|---|---|
| Specific gravity of whole blood | Male | 1.0566 |
| | Female | 1.0533 |
| Specific gravity of plasma | ..... | 1.0270 |
| Plasma protein | ..... | 6.83 Gm. per cent |
| Critical level of plasma protein | ..... | 5.5 Gm. per cent |

The specific gravity of whole blood and of blood plasma is increased in dehydration and shock. It is decreased in hemorrhage and edema.

Case I. E. M., white, male, age 70, entered the hospital with signs and symptoms of a gastric malignancy. A subtotal gastrectomy was performed on November 21, 1938. The blood studies in this case were as follows:

| Date | Sp. Gr. of Blood | Sp. Gr. of Plasma | Protein Gm. Per Cent | Edema |
|---|---|---|---|---|
| Nov. 21 | 1.0528 | 1.0241 | 5.85 | − |
| 22 | 1.0531 | 1.0222 | 5.21 | − |
| 23 | 1.0507 | 1.0230 | 5.48 | − |
| 24 | 1.0459 | 1.0217 | 5.02 | + |
| 25 | 1.0468 | 1.0225 | 5.31 | + |

On November 21, 1938 this patient had a plasma protein of 5.85 Gm. per cent. With the administration of fluids parenterally there was a sustained fall of the specific gravity of whole blood and blood plasma. Clinical edema was noted on November 24, 1938. The plasma protein as calculated from the specific gravity of plasma was 5.21 Gm. per cent (below the critical level of 5.5). The appearance of edema in this case could have been predicted at this time, forty-eight hours before it appeared clinically.

Case II. C. M., white, female, age 28, entered the hospital with extensive second and third degree burns of the face, extremities and back. Blood transfusions and fluids were given parenterally. The findings in this case were as follows:

| Date | Sp. Gr. of Blood | Sp. Gr. of Plasma | Protein Gm. Per Cent | Edema |
|---|---|---|---|---|
| Nov. 1 | 1.0485 | 1.0211 | 4.83 | + |
| 2 | 1.0457 | 1.0225 | 5.31 | + |
| 3 | Blood transfusion | | | |
| 4 | 1.0560 | 1.0278 | 7.11 | − |

Following the blood transfusions the disappearance of edema and the rise in plasma protein above the critical level is striking.

Case III. J. A., white, male, age 54, entered the hospital after several past unsuccessful operations for the relief of gastric ulcer. On October 26, 1938 a subtotal gastrectomy was performed. The blood studies after operation were as follows:

| Date | Sp. Gr. of Blood | Sp. Gr. of Plasma | Protein Gm. Per Cent | Edema |
|---|---|---|---|---|
| Nov. 1 | ..... | 1.0248 | 6.09 | − |
| 2 | ..... | 1.0249 | 6.12 | − |
| 21 | 1.0605 | 1.0261 | 6.53 | − |
| 23 | 1.0474 | 1.0245 | 5.99 | − |
| 25 | 1.0435 | 1.0226 | 5.34 | + |
| 29 | 1.0507 | 1.0264 | 6.63 | Slight |
| Dec. 7 | 1.0472 | 1.0248 | 6.09 | Slight |

This patient had a stormy postoperative course and was unable to retain anything by mouth. He soon developed a state of marked dehydration. The specific gravity of whole blood on November 21, 1938 was 1.0605 (normal sp. gr. 1.0550). With the continued ad-

ministration of fluids the specific gravity of whole blood and blood plasma fell, and on November 25, 1938, when the plasma protein fell to 5.34 Gm. per cent, edema developed. Tissue edema could have been predicted twenty-four to forty-eight hours before it appeared.

Case IV. M. DeC., white, male, age 48, entered the hospital on November 14, 1938 because of a bleeding duodenal ulcer for which he had been operated upon twice during the last fifteen years. A subtotal gastrectomy was performed on November 23, 1938. The blood findings before and after operation were as follows:

| Date | Sp. Gr. of Blood | Sp. Gr. of Plasma | Protein Gm. Per Cent | Edema |
|---|---|---|---|---|
| Nov. 21 | 1.0557 | 1.0250 | 6.15 | − |
| 23 A.M. | 1.0522 | 1.0247 | 6.05 | − |
| P.M. | 1.0552 | 1.0251 | 6.19 | − |
| 24 | 1.0541 | 1.0247 | 6.05 | − |
| 25 A.M. | 1.0494 | 1.0252 | 6.22 | − |
| P.M. | 1.0509 | 1.0245 | 5.99 | − |
| 26 | 1.0470 | 1.0230 | 5.48 | − |
| 27 | 1.0433 | 1.0231 | 5.52 | + |
| 28 | 1.0412 | 1.0217 | 5.03 | + |

The specific gravity of whole blood and blood plasma was maintained by the administration of a fairly large amount of fluid parenterally on November 23 and 24. However, a sudden fall in the specific gravity of whole blood was noted the following day. Since the fluid administered was not excessive, the sudden drop was assumed to be due to internal hemorrhage. This was apparently confirmed by the continued fall. Pitting edema appeared on November 27, 1938, when the plasma protein was 5.52 Gm. per cent.

Post-mortem examination revealed a massive internal hemorrhage from a bleeding duodenal ulcer.

Case V. A. J., white, male, age 33, was found in shock following an automobile accident. He was admitted to the hospital in coma.

Traumatic shock, caused by fractures of the femur and the skull, was diagnosed. On admission, the specific gravity of whole blood was only 1.0540, although much higher values were to have been expected. Previous investigators have shown that shock due to factors other than concealed hemorrhage is accompanied by a high specific gravity of whole blood (definitely above 1.0550). In the light of these facts, it was pointed out that the relatively low specific gravity was indicative of a massive internal hemorrhage. The patient was catheterized with the hope of detecting the source of bleeding and a massive hematuria was discovered. Four hours later the effect of the continued bleeding on the specific gravity of whole blood became even more apparent (1.0437). The aid of the specific gravity determinations made possible the diagnosis of internal hemorrhage even in the presence of coincident shock and coma.

| Date | Sp. Gr. of Blood | Sp. Gr. of Plasma | Protein Gm. Per Cent |
|---|---|---|---|
| Dec. 10 | 1.0540 | 1.0252 | 6.22 |
| 11 | 1.0438 | | |
| | 1.0437 | 1.0226 | 5.34 |
| 12 | 1.0439 | 1.0239 | 5.78 |
| 13 | 1.0475 | | |
| 15 | 1.0415 | 1.0241 | 5.85 |
| 18 | 1.0474 | | |
| 19 | 1.0467 | 1.0247 | 6.05 |

The specific gravity determinations of whole blood can also serve as a valuable aid in differentiating shock due to internal hemorrhage from that produced by other causes.

Case VI. J. B., white, male, age 42, was admitted to the hospital on November 7, 1938 with a diagnosis of perforated gastric ulcer. An operation was immediately performed and the perforation closed. The blood findings after operation were as follows:

| Date | Sp. Gr. of Blood | Sp. Gr. of Plasma | Protein Gm. Per Cent | Edema |
|---|---|---|---|---|
| Dec. 7 | 1.0582 | 1.0281 | 7.21 | − |
| 8 | 1.0623 | 1.0301 | 7.89 | − |
| 9 | 1.0588 | 1.0277 | 7.07 | − |
| 11 | 1.0520 | 1.0259 | 6.46 | − |
| 12 | 1.0523 | 1.0249 | 6.12 | − |
| 17 | 1.0557 | 1.0256 | 6.30 | − |

Case VII. R. S., white, male, age 22, was admitted with a diagnosis of second and third degree burns of the face and back. The patient had treated himself for one day with vaseline dressings and was markedly dehydrated. The blood findings were as follows:

| Date | Sp. Gr. of Blood | Sp. Gr. of Plasma | Protein Gm. Per Cent |
|---|---|---|---|
| Nov. 26..... | 1.0629 | 1.0314 | 8.34 |
| 27..... | 1.0578 | 1.0272 | 6.90 |
| 30..... | 1.0547 | | |

In Cases VI and VII the specific gravity of whole blood was increased due to dehydration. With the administration of fluids, the dehydration was overcome and the specific gravity of whole blood approached the normal.

## CONCLUSIONS

1. The Barbour and Hamilton method of determination of blood specific gravity is practical and simple.

2. By daily determinations of the specific gravity of whole blood and blood plasma preclinical edema can be determined and clinical edema predicted twenty-four to forty-eight hours before it actually appears.

3. The test serves to measure the degree of dehydration.

4. It is of value in differentiating shock from internal hemorrhage, especially in cases of coma, and is a distinct aid in following the course of internal hemorrhage.

We wish to express our thanks to Dr. S. H. Polayes of the Department of Pathology for valuable assistance in the preparation of this report.

## REFERENCES

1. Barbour, H. G. and Hamilton, W. F. *J. A. M. A.*, 88: 91, 1922.
2. Leake, C. D., Kohlm and Stebbins, G. *Am. J. Physiol.*, 81: 493, 1927.
3. Moore, N. S. and Van Slyke, D. D. *J. Clin. Investigation*, 8: 337, 1930.
4. Weech, A. A., Reeves, F. B. and Goettsch, E. *J. Biol. Chem.*, 113: 167, 1936.

# A NEW TREATMENT FOR SLOUGHING WOUNDS*

## PRELIMINARY REPORT

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THE need has long been felt for a quick-acting proteolytic agent to facilitate the early healing of wounds. We believe we have found a new chemical method which is successful in the removal of sloughs and suppuration, and which has the additional effect of promoting granulations and epithelization.

In spite of the emphasis recently placed on the ability of living tissue to combat infection, the average method of treatment seems to ignore this fundamental principle. Asepsis and antisepsis alone are not the primary requisites in wound healing. The prevention and elimination of necrosis and debris in wound deserves greater emphasis. Necrosis may be the sequel of trauma, or, more often, the effect of bacterial toxins. That necrotic tissue is a definite handicap to the healing of wounds has been amply proved. Halstead's experiment has served as a classical example: the healthy peritoneum can withstand and overcome enormously large saline suspensions of bacteria, but peritonitis will result from relatively small doses of bacteria if a piece of devitalized muscle or strangulated omentum is present in the peritoneal cavity.

If we consider the wound as a battlefield, the larger necroses can be compared with fortresses, the smaller ones with trenches of the enemy. Unless they are destroyed or eliminated, the battle cannot be carried to the open and the enemy cannot be routed. The body mobilizes its forces by the act of inflammation. By this complex process the invading bacteria are specifically assaulted and, at the same time, necrotic tissue is attacked. In fact, the identical forces act upon bacteria and necroses. In the very early stages of the struggle, the polymorphonuclear leucocytes take the lead, their number being proportional to the extent of the tissue damage. These cells not only produce a proteolytic ferment which acts upon the necroses, but actually ingest and devour dead tissue, the whole process being called phagocytosis. Within twenty-four to forty-eight hours the advance guard of blood-borne leucocytes (microphages) receives help from plasma-borne cells (macrophages), which in fact surpass the former as necrosed tissue-eaters. They nibble on the dead tissue at its free margins, forming lacunae similar to the ones produced by osteoclasts on necrotic bone. Passage of the phagocytes into the field is aided by the flushing action of the exudate, which itself helps to loosen and separate the tissues, contributing materially to elimination of dead tissue. The exudate mixed with emigrated cells and dead and liquefied tissue constitutes "pus," the "pus bonum et laudabile."

The labor of these slowly toiling scavengers is substantially supported by the process known as demarcation. Demarcation-separation of dead tissue from healthy living tissue at its borderline is entrusted to the granulation tissue. This is a proliferative change that is moving against the necrosis on all sides, quickly engulfing it and piling up a wall against it. This particular granulation tissue differs from the one destined to fill up a gap. In situations where damaged resistant tissue has to be removed, and where there must in consequence be preliminary softening, cells of a special multinucleated type are brought into the field. Such granulations in the vicinity of dead tissue, sinuses, fistulae, etc., are often exuberant in type and may grow above the

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skin level. This "wild flesh" is flabby, spongy, pale, and does not permit epithelization. Not before the wound is cleansed of all necrotic tissue do the poor granulations change to a healthy, granular, turgescent stage. Following this change, repair of lost substance and epithelization are soon effected and complete healing is surprisingly fast.

The longer it takes to rid the wound of necroses, the more granulation tissue (the progenitor of scar tissue) has to be mobilized, and consequently the more scar tissue is formed. This fact is illustrated best by a comparison of two extremes of wound healing: the minimum of scar in a wound healed by primary intention and the deeply retracted, firm scar resulting from chronic osteomyelitis or tuberculous lesions. If removal of necrosis is a physiologic prerequisite of wound healing in general, its early elimination is evidently desirable for better functional and cosmetic results.

With this exposition of the healing process in mind, we cannot fail to deplore the prevalent indiscriminate use of antiseptics.

We believe that we have an efficient proteolytic compound which (1) does not inhibit normal physiologic cleansing action; (2) stimulates normal cell division; (3) promotes granulations; (4) is by its proteolytic action unfavorable to bacterial growth (not antiseptic); (5) does not attack normal tissues; and (6) is neither toxic nor irritating. All of these properties are present in the constituents of the original formula of the late Dr. Max Baruch. This agent contains two proteolytic ingredients, namely: papain and triethanolamine. The former, an enzyme more stable than pepsin, has long been known for its digestive properties. Papain has been used as a dusting powder and also as a cream paste, but never with any degree of success, the reason being that it loses its potency within four to six hours in an aqueous medium. The difficulty is increased because it is hygroscopic, and therefore easily inactivated. The triethanolamine, although greatly inferior as a proteolytic agent, has two desirable quantities: first, it acts as a reducing agent, thereby stabilizing and activating the papain which in itself is rendered reversibly inactive by oxidation; second, it serves to moisten and thereby assists in the penetration and impregnation or hydration of protein substances by papain. Triethanolamine emulsions, because of the minuteness of their particles, further augment the therapeutic action of the lytic agents by increasing the surface area of the lytic substances.

The most efficient anhydrous solvents for the papain and triethanolamine were found by experimentation to be oleic and stearic acids and mineral oil in definite proportions in order to avoid sedimentation. Immediately before therapeutic application, water is added in the form of 5 per cent triethanolamine solution. In other words, we keep our reagents in two containers, one called "enzyme mixture" (papain, triethanolamine, oleic acid, stearic acid, mineral oil), and the other designated as "activator" (5 per cent triethanolamine aqueous solution). It is upon the compounding of the papain mixture that we rely for the therapeutic results here recorded.

Papain or caroid (papayotin, vegetable pepsin) is prepared from the juice of the fruit and leaves of carica papaya (papaw). It is an enzyme similar to pepsin but acts in acid, alkaline or neutral solution. It is a white to gray powder and is moderately hygroscopic. It dissolves about 200 times its weight of coagulated egg albumin in alkaline liquid in about five hours. It is very soluble in water and glycerine but almost insoluble in alcohol. It acts only on dead tissue. Harris in 1937 used caroid alone with greatly satisfying results in chronic suppurative otitis media. Tremble concluded that in chronic otitis media, "caroid dissolves thick tenacious mucus" and that "urea and caroid in saturated aqueous solution have a definite action on necrotic tissue. It hastens healing, has a stimulating action and is innocuous to normal tissue. In strong solution it has a slight bactericidal action which is particularly effective on

organisms which produce putrefaction. It practically obliterates all odor—dissolves pus and epithelial debris."

We have employed our method in the following manner: The stable enzyme mixture and the activator are mixed at the time of wound application by thorough stirring until the formed emulsion is of a thick milk-like consistency and appearance. The wound is cleansed with normal saline (peroxide or other oxidizing solutions being contraindicated) and is then covered with the enzyme emulsion or with gauze soaked in the emulsion; or the crevices or tracts may be injected through a cannula. Cellophane or rubber sheeting seals the area to prevent evaporation and oxidation, and to obviate any absorption by overlying dry gauze. The dressing is changed daily or every forty-eight hours depending upon the degree of discharge or necrosis present.

The results obtained in fifty-eight cases have been excellent. Our clinical material is shown in Table 1. In no case did the enzyme mixture fail to remove slough. Coincident with this result, it was noticed that the action of deodorization in foul-smelling wounds was dramatic in effect. Clean granulations appeared early, as would be expected once wound debris had been eliminated. Where enzyme applications were continued after the wounds had cleared up, rapid epithelization was evidenced. The average number of applications required was 2.34. In one case of diabetic gangrene of the foot with a large area of slough and suppuration, seven applications extending over a period of fifteen days were needed to obtain a clean granulating wound. The average time for granulations to form was 6.15 days.

TABLE 1

| Diagnosis | No. of Cases |
|---|---|
| Perirectal abscess with slough | 4 |
| Diabetic gangrene of foot | 4 |
| Electric burn of thigh—third degree | 1 |
| Carbuncle of neck | 3 |
| Carbuncle—dorsum of hand | 2 |
| Gluteal abscess—diabetic | 1 |
| Felon | 2 |
| Inguinal abscess | 2 |
| Postoperative infection with necrosis—herniorraphy | 1 |
| Postoperative infection with necrosis—umbilical hernia | 1 |
| Postoperative infection with necrosis—pilonidal cyst | 1 |
| Old leg ulcers with slough | 29 |
| Tuberculous inguinal adenitis with slough | 1 |
| Ulcer of heel—three years' duration | 1 |
| Sloughing wound of neck | 1 |
| Abscess of breast | 1 |
| Indolent wound of foot—six months' duration | 1 |
| Sloughing abdominal wound—after enterostomy | 1 |
| Decubitus ulcer over sacrum | 1 |

## DISCUSSION

The healing of wounds depends upon and is inseparably interwoven with the removal of necrotic tissue. Elimination of necrotic tissue is effected chiefly by a process of ingestion and digestion. The amount of scar tissue depends upon the length of time consumed for the removal of necrotic tissue.

## SUMMARY

1. It is our belief that the new enzyme compound used on our cases is more efficient in removing slough and in promoting the healing of wounds than any other chemical agent in general use.

2. The enzyme mixture was an efficient proteolytic agent in fifty-eight sloughing wounds.

3. Its deodorizing power and epithelium-stimulating effect was marked.

4. Its rapidity of action was outstanding.

## REFERENCES

1. HARRIS, R. A treatment of chronic purulent otitis media. *Arch. Otolaryng.*, 26: 522, 1937.

2. TREMBLE, G. E. Use of urea and caroid in chronic otitis media. *Canad. M. A. J.*, 40: 149, 1939.

# A MECHANICAL DEVICE FOR THE ADMINISTRATION OF INTRAVENOUS ANESTHETICS

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PENTOTHAL sodium has been found to be a powerful and quick-acting anesthetic agent. Its intravenous use makes it necessary that it be given slowly with only enough for relaxation of the patient, and oxygen should be administered by inhalation throughout the procedure.

Early in our use of this anesthetic agent both an anesthetist and an assistant were needed, one to administer the drug from a syringe, and the other to hold up the patient's chin and administer the oxygen. The problem of giving 20 to 30 c.c. of the solution over an hour's time was perplexing in that invariably either too much of the drug was given to prevent the needle from becoming occluded by a blood clot, or the needle would become occluded by back pressure into its lumen. The same assistant was seldom available for other operations.

FIG. 1. Illustration shows anesthetist holding up patient's chin with one hand and administering pentothal sodium from syringe by simply turning the gear wheel on the syringe holder.

The difficulty that confronted us was the administration of the drug by the anesthetist without the aid of an assistant. One of us (F.F.R) devised a simple rack and pinion gear attachment for the syringe support that solved the problem. It does away with the help of an assistant; it allows the anesthetist the use of both hands and absolute control over the amount of the drug given, and permits no back-flow into the lumen of the needle, thereby preventing occlusion.

The device as here illustrated (Figs. 1

and 2) shows the syringe held in place by a syringe clamp. The plunger of the syringe is contacted by the movable part of the rack and pinion. The anesthetic solution may be administered as slowly as desired by simply turning the gear wheel with the thumb and index finger, which is geared about the same as the low power on a microscope.

During the past eight months approximately 1000 anesthetics varying in time from ten minutes to more than two hours have been given with complete satisfaction by this method.

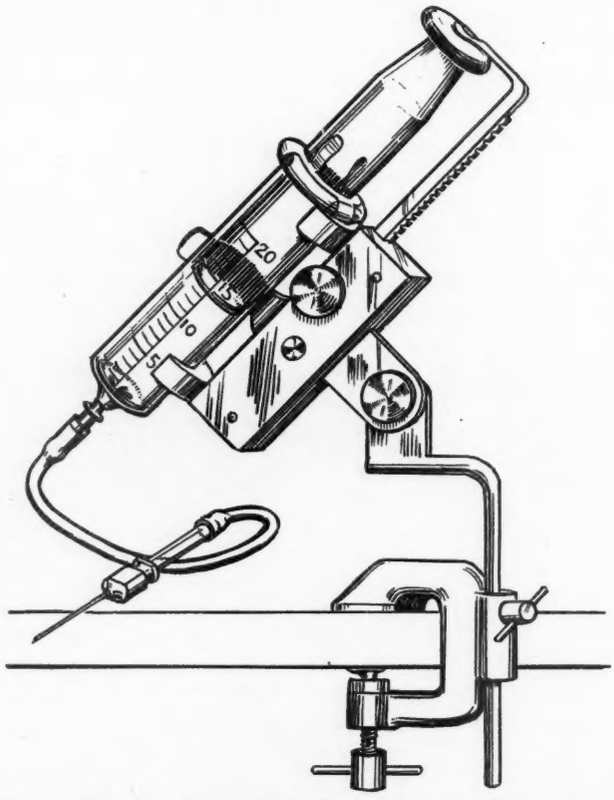


Fig. 2. Drawing shows detailed construction of apparatus.

# A SIMPLE METHOD FOR THE COLLECTION OF BILE FROM T TUBES*

JOHN H. GRINDLAY, M.D.

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ROCHESTER, MINNESOTA

THE small detail of an apparatus in which to collect bile from draining T tubes is one which is not likely to receive much attention. The patient with a T tube is usually encumbered with a device, such as a small glass bottle or hot water bag, which is poorly adapted to the purpose and is awkward for the patient to wear.

A large, rubber balloon, connected to the T tube by means of a special glass adapter, has been found to be a satisfactory, practical and comfortable apparatus for collecting bile from patients on whom† choledochostomy has been performed. (Figs. 1 and 2.) This two-piece apparatus is light in weight, can be worn comfortably and inconspicuously under clothing, does not permit spilling of bile, provides for direct observation of the bile through the glass connector, and is inexpensive. In addition, contamination of the collected bile can be prevented by simple precautions.

The rubber balloon which is used measures 5 by 6 inches (12.5 by 13 cm.), has a fluid capacity of 300 c.c. without stretching, and is made of white rubber, heavier than that commonly used for toy balloons. The balloon can be boiled or autoclaved many times. The funnel-shaped glass connector is made of pyrex glass. The small end is shaped like an ordinary glass connector and is slightly greater in diameter than the usual T tube. A standard connector diameter has been found satisfactory for all ordinary sizes of T tubes. The large end of the connector is slightly larger in diameter than the mouth of the balloon. A deep, narrow constriction of this end of the connector serves to hold the reinforced lip of the balloon tightly in place.

FIG. 1. Apparatus for the collection of bile.

The balloon and glass connector are boiled or autoclaved before being fitted together. We have used sterile gloves and a sterile towel in stretching the balloon mouth over the large end, and the T tube over the small end of the connector. When it is necessary to empty the balloon, the T tube is disconnected from the glass connector, but first, the junction is thoroughly cleansed with 70 per cent alcohol. Inverting the balloon then serves to empty it completely and quickly through the connector. The same apparatus can then be reapplied without contamination of the parts exposed to bile.

† The drainage balloons may be obtained from the Sterling Rubber Company, 176 North Wacker Drive, Chicago Illinois (catalogue #16P).

* From the Institute of Experimental Medicine, The Mayo Foundation, and from the surgical service of Dr. Waltman Walters.

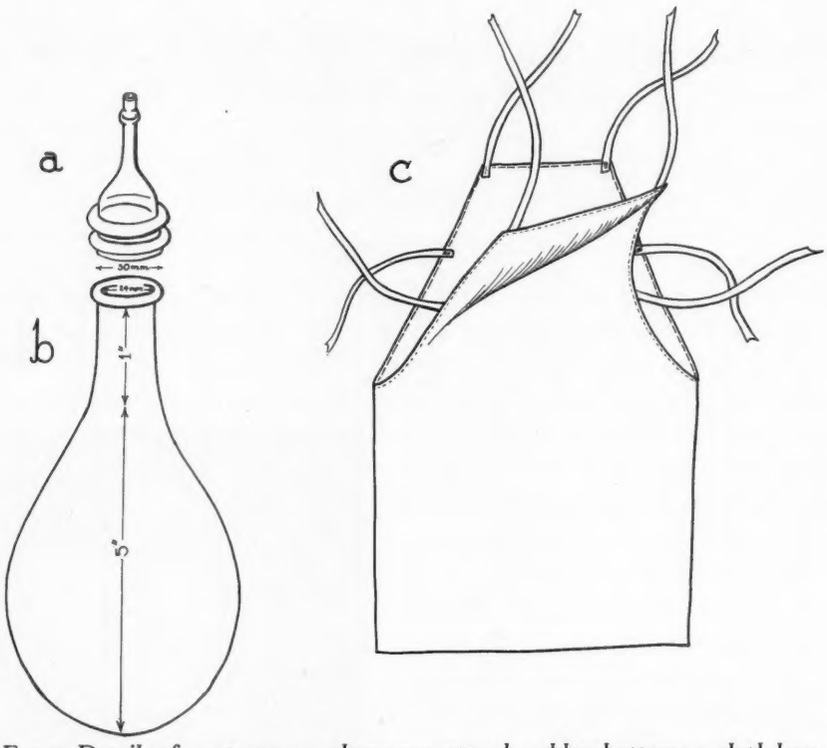


FIG. 2. Details of apparatus: *a*, glass connector; *b*, rubber bottom; *c*, cloth bag.

A washable cloth bag that fits around the balloon is used to contain the apparatus and is supplied with ties that serve the dual purpose of closing the bag and of fastening it to a binder worn by the patient.

Other uses for which the apparatus has been successfully employed are the collection of bile from cholecystostomy tubes and urine from nephrostomy or ureterostomy catheters.

# THE CAUSES FOR FAILURE IN THORACOPLASTY*†

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THE purpose of this paper is to state certain facts regarding the causes of failure in extrapleural thoracoplasty which have come to our attention in a retrospective analysis of our cases from 1931 to 1939. During the early years of our work it was our custom to do an incomplete, very selective thoracoplasty both as to number and extent of ribs removed over the cavity-bearing area, releasing the apex of the lung as in an apicolysis or extrapleural pneumothorax, and to depend upon extensive gauze packing or inflated rubber balloons to maintain compression. Within the first several weeks the results were good; the sputum became negative and roentgenologically the cavity or cavities were closed. However, removal of the gauze packing or rubber balloon left a large dead space into which the lung reëxpanded; the cavity or cavities reopened, the sputum became positive. The final results were failures.

Furthermore, many of these wounds became infected. There was an extensive and intensive parietal pleural reaction, which caused the pleura and the tissues of the chest wall to become thickened, fibrocartilaginous in nature, with frequent calcification. The result was immobilization of the lung in the reëxpanded position. Since no further surgical procedures could be carried out in the presence of infection it was necessary to wait weeks and often months for these large infected wounds to clear up, and when second and third stages or the revision thoracoplasty were carried out, practically nothing could be accomplished.

As a result of these experiences, we have given up the use of foreign substances for compression. It is interesting to note, however, that in two cases in which the gauze packing was accidentally left in the chest wall no ill effects or untoward symptoms have been seen. The sputum has remained negative, and roentgenologically the huge cavities have remained closed.

No doubt it would be a great aid in thoracoplasty if some neutral substance could be left permanently in the chest wall to maintain constant compression, but after our ill-fated experiences with various substances, such as gauze, rubber balloons, crisco, etc., we prefer that some other group carry on with these experiments—we are cured.

Our results also were comparatively poor in cases where an extrapleural thoracoplasty was done in the presence of pneumothorax or pneumothorax with effusion, regardless of the extent of the surgical procedure. The immediate results appeared to be good, but when the pleural effusion or pneumothorax was absorbed the lung reëxpanded and the cavity or cavities reopened. Here again the pleural reaction was marked, causing a parietal pleural thickening, and, despite a revision thoracoplasty, very little was accomplished. Consequently no surgical procedure is carried out at present in the presence of pneumothorax or pneumothorax with effusion, until the portion of the lung to be collapsed is fully reëxpanded.

We have also concluded that many patients should have been operated upon months or even years earlier than was actually the case, rather than to have been left on bed rest and observation for long periods of time. In some of these cases our surgical results were not good. In these cases unquestionably certain inherent path-

* Read before the clinical session, Tuberculosis Sanatorium Conference of Metropolitan New York.

† From the Hudson County Tuberculosis Hospital and Sanatorium, Dr. B. S. Pollak, Medical Director.

ologic changes had taken place in the lung, the parietal and pulmonary pleura, and the chest wall itself, which precluded any effective collapse.

In still other cases, phrenectomy was performed. This did not close the cavity and valuable time was lost before thoracoplasty was done.

It was found also in cases where ineffectual pneumothorax had been carried out for a long period of time, that the parietal pleura had become thickened and there was immobility of the chest wall to a varying degree. The results of extrapleural thoracoplasty in these cases were not so good as in cases where ineffectual pneumothorax had been carried out only for a short period of months, or in cases in which pneumothorax had not been done at all.

We feel now that cases should be observed for a comparatively short period of time. Long periods of observation and of ineffectual pneumothorax, especially in cases of adherent, peripheral apical cavities, are inadvisable. These patients should come to surgery sooner.

In our surgery per se we are of the opinion that we have been too conservative—that we have been too limited and too selective in our surgical procedure both as to the number of and extent of ribs removed. In our procedures the apex of the lung was freed as in the Semb's operation; this caused a vertical drop and a vertical collapse of the cavity. This also, very often, caused the cavity to drop an interspace or more without actually collapsing the cavity—it merely changed its shape. Nor were we very thorough in the juxtavertebral resection of the ribs or the transverse processes which should aid in the obliteration of the cavities throughout the greater diminution of the transverse diameter of the cavity.

Our tendency was to delay too long between stages, hoping that thoracic contraction would occur and thus obliterate cavities. This, however, did not occur in a large majority of cases. Owing to this delay, in many cases the ribs regenerated, the pleura and the tissues of the chest wall became fibrocartilaginous, while in others calcification occurred. Therefore when the next stage operation or a revision thoracoplasty was done, the patient was subjected to a difficult, bloody, shocking procedure. A complete, effectual, extrapleural thoracoplasty with a maximum delay of four weeks between stages would have accomplished our aim in a majority of the cases.

As a result of our too limited and too selective thoracoplasties some of the cases that at present show negative sputum and roentgenologically closed cavities, are likely at some future date to show a positive sputum with reopening of the cavities. Some will develop, as they have in the past, a contralateral spread.

Although the amount of collapse which will follow any operative procedure is unknown, we do know that some cases will show a much greater degree of thoracic contraction after thoracoplasty than others. Nevertheless our feeling is that we should be more radical with respect to number and extent of ribs to be removed, with especial effort to eliminate the paravertebral gutter and to shorten the interval between stages.

It is difficult to compare statistically the results of different clinics, because of the wide variations in the interpretations of operative indications. Despite all our shortcomings, 58 per cent of our cases after three to five years have absolute cavity closure by Roentgen determination, and negative sputum. The immediate postoperative results showed a much higher percentage of closure.

## CONCLUSIONS

Pulmonary tuberculosis is neither a medical nor a surgical disease, but rather one in which all specialties may add something to the patient's chances of recovery. The results of thoracoplasty depend not only upon the surgeon (granting that an effectual, complete, selective type of extrapleural thoracoplasty is performed), but upon the coöperation of other specialists.

# A NEW AND EFFECTIVE ANORECTAL DRESSING*

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FOLLOWING anorectal operations bacteria-laden moisture continuously bathes the operative field. This irritating excretion promotes profuse granulation, retards expithelization, induces painful wounds, creates a sense of uncleanliness, excites pruritus ani, slows the process of healing, and leads to the development of a tough, deforming and sensitive scar. Postoperative dressing with an ointment or powder markedly increases this moisture.

It occurred to us to seek an analgesic, astringent, germicidal and mucilaginous dressing. The latter property causes prolonged contact with the tissues. Following experimentation with many agents, we selected a mixture of 3 per cent gentian violet, 10 per cent tannic acid and 1 per cent pontocaine in a heavy mucilage of acacia.

To prevent the formation of crystals and small lumps, this mixture must be painstakingly compounded. A thick and smooth mucilage is essential. The following plan for preparation of 500 c.c. of the mixture is recommended: Dissolve 5 Gm. (50 tablets, each gr. 1½) of pontocaine in 4 ounces of distilled water. Add 50 Gm. of tannic acid and stir until dissolved. Add 15 Gm. of gentian violet crystals and stir until a solution is effected. Strain through gauze into a large mortar. Add slowly, while agitating vigorously, sufficient acacia to make a thick paste. This requires approximately 250 Gm. of acacia. Now add gradually, stirring continuously, distilled water to make 500 c.c. Let the mixture stand in mortar twenty-four hours, stirring every three or four hours if possible. At the end of this time agitate vigorously with pestle, then force the thick mucilage through two layers of gauze with the aid of a spatula. This mixture remains stable when kept tightly corked.

During the first thirty-six hours following completion of the dressing, a light foam may form on its surface, precipitating a thin layer of acacia. The acacia will return into solution if stirred. To facilitate its application on the gloved finger or on an applicator this dressing should be dispensed in a wide-mouthed container. A properly made mixture spreads on the skin as a smooth and homogeneous mucilage, drying quickly to remain as a purple paint.

When an operation is concluded, we apply the dressing generously to all wounds: circumanal, anal and rectal. Subsequently the operative field is dried after each sitz-bath and an attendant then applies the dressing to the circumanal wounds. During the first postoperative week the dressing is applied twice daily to the wounds within the anal canal and rectum by means of a small, soft, mop-like applicator. Thereafter, its application within the canal is made once daily by us and to the circumanal wounds by the patient or an attendant after each sitz-bath.

We have employed this dressing routinely and with uniform results since January, 1938. Its proved advantages are: (1) absence of postoperative oozing; (2) marked control of postoperative hyperesthesia, pain, edema and pruritus ani; (3) a mild tanning of the tissues with continuous dryness of the field; (4) absence of exuberant granulation. This tissue remains firm and level with the skin, presenting a healthy pink appearance without a tendency to bleed, and (5) rapid healing with resultant thin and flexible scars.

* Read before American Proctologic Society, Brooklyn, New York, June 25–27, 1939.

# A METHOD OF HOLDING MULTIPLE ARTERY CLAMPS

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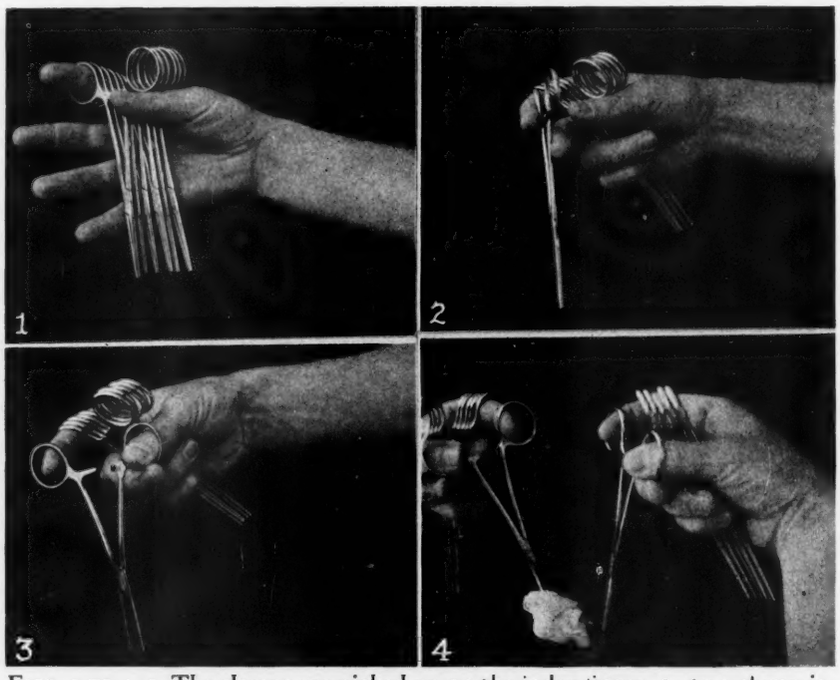
AN artery clamp can be held like a scissors or between the thumb and forefinger like a thumb forceps. (Fig. 3.) Holding it the latter way, one has better hand to point coördination for this is the natural manner in which one holds other small hand tools such as a pencil, pen, brush and scalpel. All parts of the wound can be reached without twisting the upper arm or body. Either hand can be used with dexterity. It is also much the handier method if the team works sitting down. The clamps can be passed one at a time, or if one is working without an instrument nurse, six clamps can be taken up in a bunch.

Pick up the clamps by passing finger one through the upper ring. (Fig. 1.) Hold the shafts close in the palm with fingers three and four. The tips of the other fingers remain free for retracting or sponging. Or the sponge can be held with the clamp in the other hand. (Fig. 4.) When a clamp is needed, flex fingers two, three and four behind the next clamp. Grasp this clamp firmly between fingers one and two (Fig. 2) and push it forward. Then flex these two fingers and bring the ring of the clamp over the end of the thumb. Grip the thumb ring tightly between the thumb and finger two. (Fig. 3.) Unlock the instrument by slightly apposing the thumb and index finger. The open clamp is now held poised for grasping, gripped only by the thumb and finger two. The index finger does not grip but opens and closes the jaws and locks them after the vessel has been grasped.

When the clamps are picked up for tying off, they can be held in this same manner and unlocked and removed.




FIGS. 1 TO 4. 1, The clamps are picked up on the index finger. 2, As a clamp is needed, it is pushed forward and grasped between the thumb and second finger. 3, Clamp unlocked and poised for grasping a bleeder. 4, The assistant may hold clamps in both hands.

# CASE REPORTS

## THECOMA OF THE OVARY

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THE origin of tumors of the granulosa and theca cells of the ovary is still a matter of debate. It is not even certain that these tumors are two distinct entities. For that reason, it seems wise to publish any cases that may serve to clear up the problem.

The case under discussion was a fibromatous tumor of the right ovary. The tumor extract contained a substance whose reaction was similar biologically to estrogen, that is, it caused an enlargement of the uterus in the mouse test.

The patient was an unmarried woman of 36 whose menstrual flow had always been regular. Following intercourse, there was no menstrual period for six weeks. Then there was slight bleeding, followed by scant bleeding at irregular intervals for six to eight weeks. At the same time the patient became aware of an enlargement of the uterus. Believing that she had become pregnant and that an incipient abortion was in progress, she consulted the writer.

The uterus was enlarged to the size of a four months' pregnancy; however, it was not so soft as a pregnant uterus and Hegar's sign was not marked. The right ovary was enlarged to the size of a fist. The Aschheim-Zondek test was done and returned as negative. Accordingly, a diagnosis of granulosa cell tumor was made, and the neoplasm removed by laparotomy.

Macroscopically, the tumor resembled a fibroma with distinct lighter nodules varying in size from that of a pinhead to that of a cherry and elevated from the surface. Microscopically, the picture was that of a fibroma with strands of connective tissue interlacing. In the interstices, however, were masses of oval and polygonal cells whose nuclei stained deeply and whose protoplasm showed vacuoles and, under fat stains, proved to be lipoids.

### DISCUSSION

The Wilson Laboratories prepared an extract of the tumor to ascertain its endocrine properties. The extract was injected subcutaneously into infantile mice in equal quantities for four successive days. At the same time infantile mice were treated with theelin and anterior pituitary hormone. Mice of the same litter were kept as controls under identical conditions. The uteri of the animals treated with the tumor extract, as well as those treated with theelin, showed a distinct enlargement as compared with the controls; there was no reaction on the ovary. The reaction was, therefore, that of estrogen and this estrogen was obtained from a fibromatous tumor. It may well be assumed that the estrogen did not come from the connective tissue but rather from the vacuolized cells. Nothing seems to speak for the assumption that the hormone was formed outside the tumor and only stored up in it, because the patient had at no time been pregnant.

The ability to produce female sex hormone is common to a granulosa cell tumor and to the tumor under discussion, which may be tentatively classed as a thecoma. Both produce estrogen and cause glandular cystic hyperplasia of the endometrium. However, macroscopically the granulosa cell tumors are soft formations surrounded by capsules of connective tissue. They can acquire considerable size (the tumor herein

described weighed 7 pounds); the interior contains a spongelike substance which, as the tumor increases, inclines to disintegrate to form cavities that contain a blood-tinged fluid. Theca cell tumors are hard and show a preponderance of connective tissue in which the tumor cells proper are arranged in groups. Microscopically both tumors are composed of connective tissue and tumor cells. In granulosa cell tumors the tumor cells predominate, whereas in the thecomas the tumor cells are much fewer. It would seem that the granulosa and theca cells have the power to multiply and grow at the expense of the connective tissue. In the case of granulosa cell tumors the cells not only multiply, but the tumors show progress from trabecular to solid and follicular form.

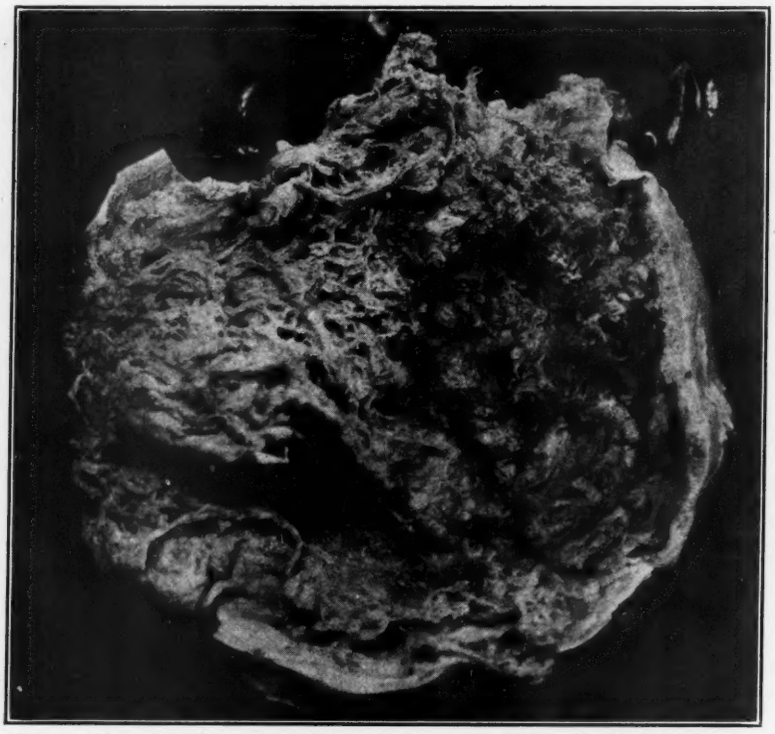

Fig. 1. Cross section of granulosa cell tumor. (*Am. J. Obst. & Gynec.*, 36: 688–693, 1938.)

The granulosa cells differ from the theca cells in various properties; the former have weakly defined cell outlines, their nuclei stain well, are of good size and are round or oval, and it is very seldom that any lipoid substance is found in these cells.

According to Motta, the cells of the corpus luteum and of the luteinized granulosa cell tumors contain a preponderance of simple breaking lipoids, whereas the lutein cells of the theca tumors show a preponderance of double breaking lipoids; however, these cells lose their double breaking property when they are slightly warmed. The presence of lipoid substance in both kinds of cells is evidence that in some respects these tumors are related. These reasons, together with the morphologic ones, argue for a common origin of granulosa cell and theca cells from the mesenchyme of the coeloma, as Fischel and Schiller maintain, but the stimulus transforming the mesenchyme cells to granulosa cells is apparently different from the one which calls forth the formation of theca cells.

According to Hertwig-Waldeyer, the parenchymal cells of the ovary develop by ingrowth of the gonadel surface epithelium into the underlying embryonic connective tissue. From this point of view we should have to assume that such highly differentiated cells as ova and spermatozoa could develop out of mesoderm cells which originally cannot be distinguished from the cells

which supply the endothelium of the peritoneum. This appears unlikely, as does the opinion that the sex cells originate in the epithelioid elements of the germ gland at a later stage of the development.

After the work of Nussbaum, Galton, Boveri and Beard, Weissman's theory is justified, that the sexual cells follow a course of development which differs altogether from that of the other somatic cells. They apparently retain their totipotent quality, which is essential to the first offshoots of the fertilized ovum. The sexual cells differ at an early stage from other embryo cells by reason of the chromatic elements of the nucleus. Their progress can be followed through the earliest segmentation cells along a definite course. In mammals they take their way into the primitive streak, then into the dorsal wall of the intestine; then they wander through the connective tissue of the mesenterium toward that spot on the dorsal wall of the body where the germ gland arises. So primitive sexual cells (archygonocytes) seem to be the mother cells of sperm cell and ovum. The latter are not derivatives of the germ epithelium nor the product of the sex glands, but direct derivatives of the fertilized ovum.

The difficulties in explaining the histology of granulosa and theca cell tumors are easier to understand if we accept Fischel's view that the granulosa cell originates in the mesenchyme through differentiation called forth by the formative stimulus of the primitive sexual cells (archygonocytes), after the latter have made their distant migration to the spot. And the theca cells are apparently cortex cells of the ovary modified by the ripening process of the

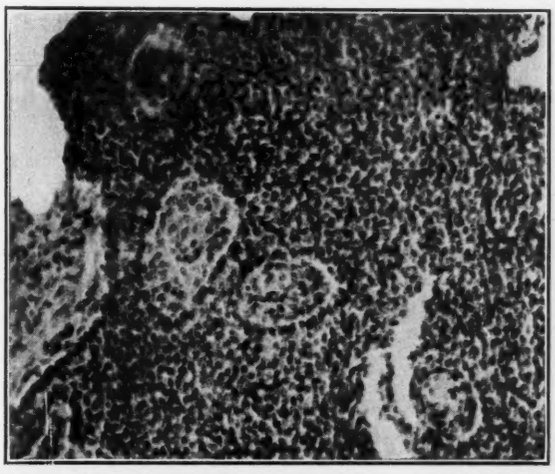

Fig. 2. Granulosa cell tumor, solid mass and follicle formation. (*Am. J. Obst. & Gynec.*, 36: 688–693, 1938.)

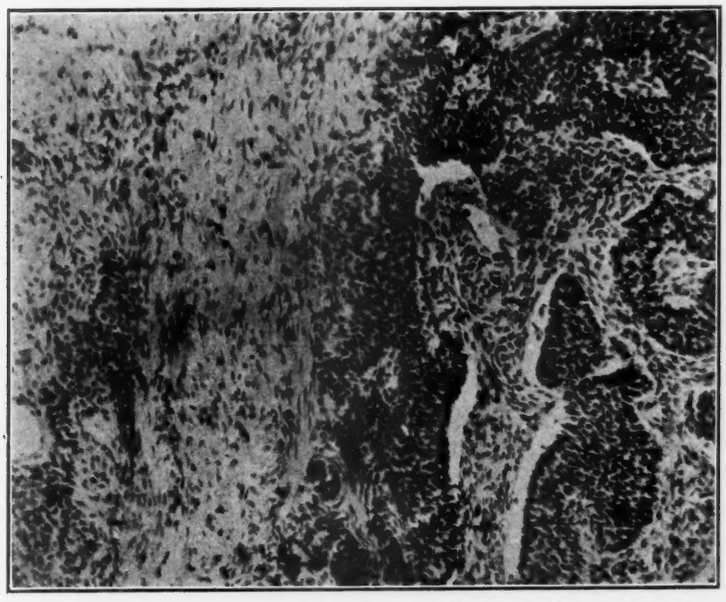

Fig. 3. Granulosa cell tumor, trabecula formation. (*Am. J. Obst. & Gynec.*, 36: 688–693.)

Graafian follicle. "They are not a part of the follicle, take no part in formation of the corpus albicans" (Gardner).

FIG. 4. Thecoma: fibroma with nodules of theca cells.

These cells seem to be analogous to the decidua cells of the endometrium (decidua mentrualis and graviditatis). In the atretic cysts there is no intermingling of granulosa and theca cells. The theca cells are apparently the result of a softening process of the cortex to facilitate the rupture of the follicles.

Tumors of desmoid nature containing lipoid were first differentiated by Loeffler and Priesel who called them "fibroma thecacellulare xanthomatodes ovarii." These tumors have been separated from the granulosa cell tumors in the opinion of some investigators, while others oppose their segregation because granulosa cell tumors and thecomas have a common origin from the embryonic ovarian mesenchyma. This is true and is in accordance with Fischel's view that the mesenchymal core of the plica genitalis undergoes its transformation under the influence of the primitive ovum (archygonocyte); the archygonocyte causes the development of granulosa cells and the ripening of the ovum transforms the adjacent connective tissue into theca cells similar to the transformation of connective tissue cells in the uterus into decidua.

## SUMMARY AND CONCLUSIONS

1. An ovarian tumor is reported which macroscopically and microscopically is classified as thecoma, a fibroid tumor containing nodules of theca cells of different size.

2. The tumor produced estrogen.

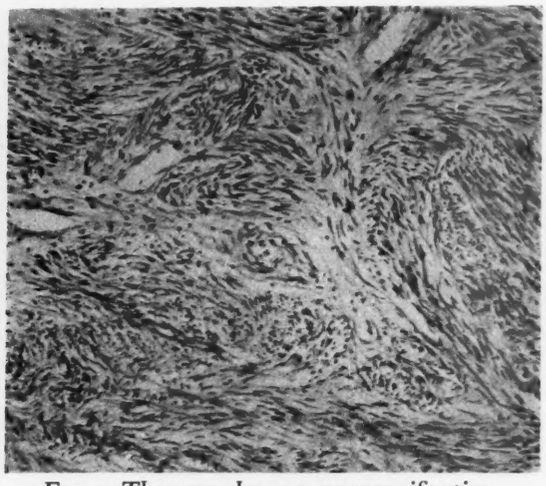

FIG. 5. Thecoma: low power magnification.

3. It caused a glandular cystic hyperplasia of the endometrium, enlargement of the uterus and irregular metrorrhagias.

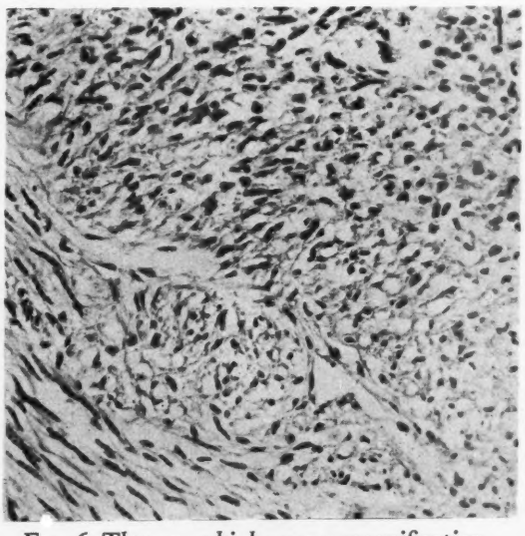

FIG. 6. Thecoma: high power magnification.

4. In spite of similarities in granulosa cell and theca cell tumors, i.e., estrogen production and lipoid contents, there are differences macroscopically and microscopically.

5. A segregation of granulosa cell tumors and thecomas seems to be justified.

## REFERENCES

1. GREENHILL, J. P. and GREENBLATT, R. B. Status of the thecoma and its relationship to the granulosa cell tumor. *Am. J. Obst. & Gynec.*, 36: 684, 1938.

2. MELENIK, PERRY and KANTER, A. E. Theca cell tumors of the ovary. *Am. J. Obst. & Gynec.*, 27: 41–51, 1934.
3. DAMM, P. N. Menstruationsstoerungen Hormonal en Ursprunges. Kopenhagen, 1936. Levin and Munksgaard.
4. MOTTA, G. Genese des Corpus Luteum und der Granulosa des Follikels. *Ztscbr. f. Gynäk.*, 60: 1547, 1936.
5. GARDNER, W. S. Normal and pathologic developments from the cells lining the graafian follicle. *Surg., Gynec. & Obst.*, 67: 455–466, 1938.
6. GEIST, S. Theca cell tumors. *Am. J. Obst. & Gynec.*, 30: 480–495, 1935.
7. LOEFFLER, E., and PRIESEL, A. Theca cell Tumoren. *Beitr. z. path. Anat. u. z. allg. Path.*, 90: 199, 1932.
8. TRAUT, H. F. and BUTTERWORTH, J. S. The theca, granulosa, lutein cell tumors of the human ovary. *Am. J. Obst. & Gynec.*, 34: 987, 1937.

TUMOURS form occasionally in the root of the mesentery and behind it, constituting the retroperitoneal lipoma or sarcoma. . . . It is seldom that a retroperitoneal sarcoma can be enucleated, and treatment by irradiation must be employed.

# RARE MALFORMATION OF THE ARM*

## DOUBLE HUMERUS WITH THREE HANDS AND SIXTEEN FINGERS

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WHITE PLAINS, NEW YORK

ALTHOUGH the literature of the past 150 years abounds with case reports of supernumerary members, we have been unable to locate any report of an aberration such as is here presented. In this case a woman of 52 had gone through life with a normal left arm, but a right arm double average size and terminating in three hands of more or less normal appearance.

Paré reports an almost legendary monster born with four legs of equal proportions and two supernumerary arms the day peace was made between the Venetians and the Genoese. Haller[1] reported several instances of supernumerary extremities in 1758, and Saviard[1] saw an infant in 1867 which had ten toes on each foot and ten fingers on each hand. A patient cited by Murray[2] came closer to the type of anomaly seen in our case: She had an abnormal left arm with imperfect flexion of the elbow, which terminated in a double hand with rudimentary thumbs and webbing of the middle and ring fingers.

Bradford and Lovett[3] saw a patient with fifteen fingers and thirteen toes. Joachimstal[4] and Klaussner[5] also reported similar disturbances. The latter in 1900 described double humeri. The most recent monographs which deal with deformities of the upper extremity are those of Mueller[6] and Gruber.[7] Apparently none of these authors had observed an anomaly so bizarre as occurred in the present case.

### CASE REPORT

E. G., an unmarried woman of 52, accidentally came under our observation. She had as her sole deformity an amazing right arm, double the size of the left. The lower portion of this arm was imperfectly separated into two parts by a shallow skin fold which ended at the elbow. The forearm terminated in three hands, a proximal, a distal and a middle, which we may call hands *A*, *B*, and *C*. In addition there was what might be considered a rudimentary fourth hand which consisted of supernumerary fingers and a large carpal bone. (Figs. 1 and 2.)

The proximal (upper, cranial or anterior) hand, *A*, was kept in maximal pronation. The lower, distal, caudal or posterior hand, *B*, was kept in maximal supination. The middle or lateral hand, *C*, was maintained in a position of hyperpronation. *C* was attached to *A* by a web formation at the ulnar side, in the center of which the two rudimentary finger stumps could be seen. *C* was likewise attached to *B* by a common thumb which served them both. The carpal bases of all three hands were inserted in a kind of funnel arrangement, so that each hand was in a separate plane, rotated 90 degrees from the others.

The three hands could perform useful tasks alone or in combination, since they had muscle groups of normal shape and number. The radial artery could be palpated at all three radial areas. The hands could be flexed and extended independently, making all possible combinations of flexion and extension possible. However, for obvious reasons, the patient had found it advisable to cover two of the hands with a scarf arrangement, using only one, *B*, the lower hand for her work. For twenty-five years she had filled a position as telephone operator and file clerk, apparently without major inconvenience. She relied chiefly on her left hand, using the *B* hand on the right side for such activities as a left-handed person might.

The family history, so far as the patient or her 83 year old father could remember, was completely negative. The patient's own growth and development had been in all other respects normal. Whether as a result of the deformity or by normal temperamental inclination, the pa-

* Presented at the New York Academy of Medicine at the joint meeting of the New York and Philadelphia Orthopedic Sections.

tient was shy, secretive and introverted. She tried to evade any inquiry about or examination of her arm and resisted any offers to attempt its correction.

fourth cervical vertebra at a sharp angle. The spina scapulae could not be palpated. The cervicodorsal spine showed marked curvature to the right.

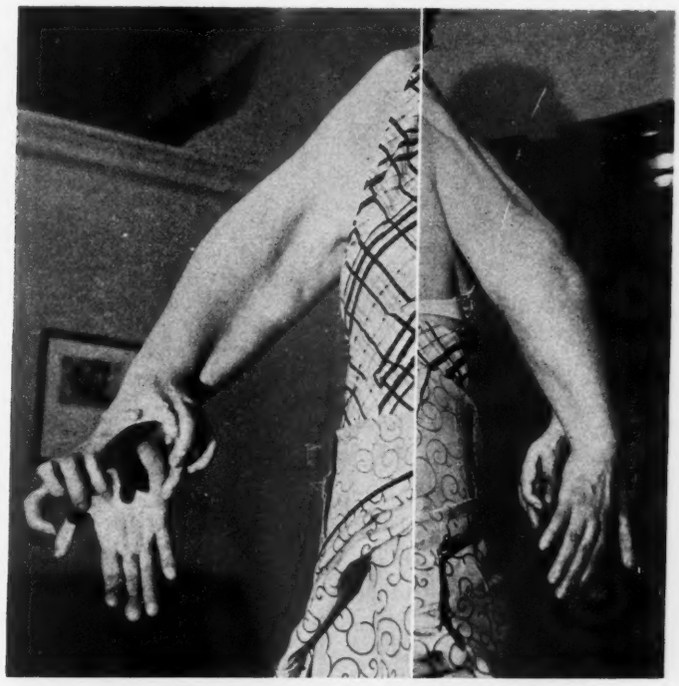

Fig. 1. Fig. 2.

Fig. 1. Anterior view with forearm in supination. Note common thumb for distal and middle hands; also rudimentary finger in web between middle and proximal hands.

Fig. 2. Posterior view—proximal and distal hands visible.

The abnormality, present of course from birth, had never troubled her. The abnormal size of the arm and shoulder had induced her to have a shoulder brace made when she was 25 years old. This was intended to keep the shoulder blade back; but since it caused more discomfort than the disability it was designed to help, it was soon discarded.

*Examination.* The arm was maintained in 20 degree abduction, but could be abducted to 45 degrees at the shoulder. Adduction of 20 degrees, flexion, forward and backward rotation and retroversion of 50 degrees were also possible. Outward rotation (with the arm to the side) was the full range of normal, but inward rotation stopped at two-thirds of the normal range.

The combined elbow showed a flexion contracture of 20 degrees, could be flexed to 45 degrees, supinated normally, but pronated only halfway. The shoulder blade was 9 inches long and 9 inches across (as compared with 6 by 5 inches for the left shoulder). It joined the

The acromion on the right side was 3 inches higher than on the left, due to a giant scapula. The sterno-acromial line was anteverted and uplifted (5.2 inches long at the right side and 6.2 inches at the left).

Hand *A* (proximal) was kept in 10 degrees dorsiflexion, its distal phalanges flexed at an angle of 10 degrees, with the thumb in 45 degree adduction under the middle hand. Each finger could be moved separately in extension and flexion. A fist could be made and the patient could hold a fountain pen with this hand. However, the thumb, due to a flexion-adduction contracture, had little power of abduction, adduction and opposition.

Hand *B* (posterior or caudal) was the most normal in appearance of the three, and it was this one which the patient was accustomed to keep uncovered for use. Its thumb, however, was common also to hand *C*, which formed the lateral plane of the hand triangle. This thumb had full power of adduction and opposition.

In hand *C* a flexion contracture of the middle phalanges of the third, fourth and fifth fingers was noted. The range of motion of the three hands is indicated in Table 1.

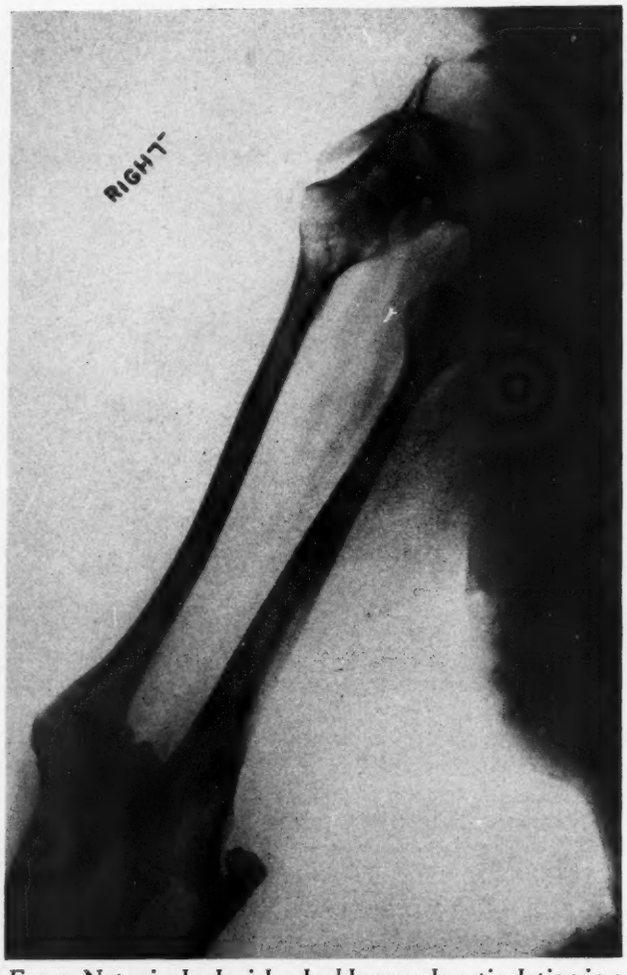


FIG. 3. Note single clavicle; double scapula articulation in a giant scapula; double humeri.

*Roentgenographic Examination.* *X*-rays revealed two humeri of normal length and shape.

TABLE 1

| | Hand A Degrees | Hand B Degrees | Hand C Degrees |
|---|---|---|---|
| Wrist flexion | 10 | 10 | 20 |
| Wrist extension | 10 | 10 | 10 |
| Abduction | partial | 20 | 10 |
| Adduction | partial | 20 | 10 |
| Basal phalanges (flexion) | 70 | 80 | 90 |
| Middle phalanges (flexion) | 30 | 45 | 10 |
| Distal phalanges (flexion) | 10 | 10 | 10 |
| Interossei | normal | normal | normal |

(Figs. 3 and 4.) Both humeral heads articulated in separate shallow glenoid fossae of a giant scapula. The scapula extended from the seventh rib (its mesial border 2½ inches from the right transverse dorsal processes); the upper end converged up towards the fifth cervical vertebra. The latter showed evidence of spina bifida.

The clavicle, originating at the level of the middle of the fourth rib, led to a bulky acromion near the outer humerus. No second clavicle was visible, but a large coracoid process, 6 inches long, could be seen at the mesial side of the inner humeral head, reaching half an inch below the inner side of the surgical neck. At the articulation of the inner humerus, a shallow acetabulum articulated only halfway with the mesial part of the inner humeral head. No acromion was seen. An irregular jagged border led to the second, outer, humeral articulation, with no formal glenoid. Only one acromioclavicular joint could be distinguished with markedly enlarged acromion embracing the humeral head laterally. Three sharply defined

bony edges led to the upper lateral scapula angle. The scapula was markedly shifted to the lateral chest wall, only one-eighth of its width being in contact with the ribs. It was very much rotated outward.

Fig. 4. Showing relationship of the forearm bones at the elbows and wrists.

The humeri showed extremely flat trochleae which articulated incompletely and irregularly with the semilunar notches of the ulnae. The ulna for hand *A* was proximally and medially displaced. The radial head was opposite the inner humerus and showed marked flattening. (Fig. 5.) Two radii and three ulnae were present in all. The inner radius was normal in appearance and articulated with a shallow trochlea of the inner humerus. Corresponding to it was a medial ulna in only partial articulation with the medial condyle. The coronoid process and the olecranon were not in contact with the coronoid and olecranon fossa, these being absent, but articulated with an incomplete carpal row of hand *B*. Lateral to the radius a single ulna was attached to the lateral condyle of the medial humerus. Between the two ends of the humeri, without joint contact, the olecranon and coronoid processes were markedly elongated, the distal end articulating with the proximal carpal row of hand *C*. The radius corresponding to this ulna was missing. (Fig. 7.) The third ulna articulated normally with the lateral humerus. However, the medial condyle was also enlarged and elongated. The proximal end of the ulna was covered by an elongated radius without capitellum formation. It was only loosely attached to the lateral epicondyle. This radius and ulna articulated with hand *A*. The coronoid process, however, apparently pointed backward, while the olecranon articulated in front of the humerus. This whole humeroulnar mech-

anism apparently had rotated 180 degrees. The dimensions of these parts are detailed in Table II.

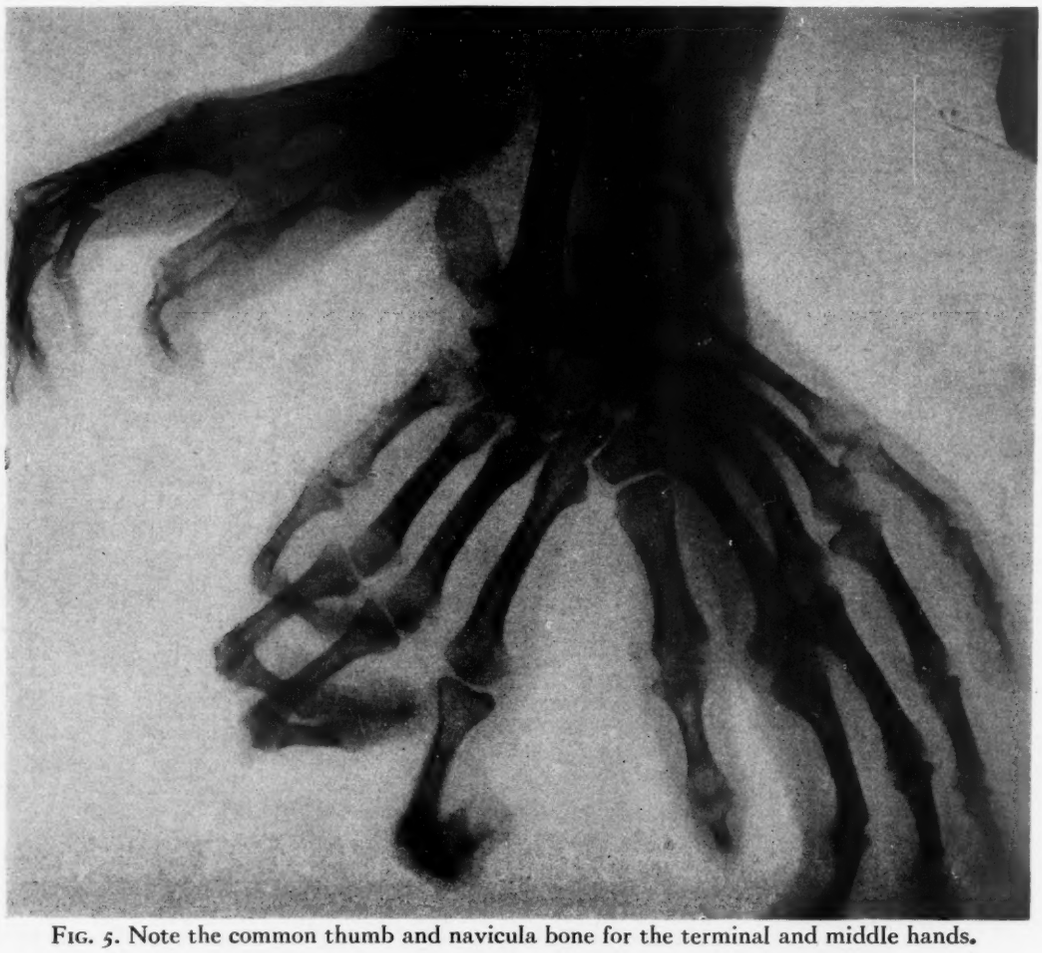

FIG. 5. Note the common thumb and navicula bone for the terminal and middle hands.

The hands themselves made an interesting *x*-ray picture. The upper, cranial hand, *A*, showed incompleteness in the form and number of the carpal bones, but had phalanges gracefully slender and normally shaped. This hand was maintained in an 80 degree dorsiflexion, complete pronation, with the middle and distal phalanges flexed. The lower hand, *B*, was complete in respect to carpal bones and its phalanges were entirely normal. The thumb, as previously noted, served hands *B* and *C* in common. Hand *B* was in complete supination. *A* and *B* had both radius and ulna, while *C*, kept in midpronation and supination, had only an ulna, maintaining connection with the radius of *B* through the radius, common scaphoid and trapezium. The carpal bones of *C* were normal.

TABLE II

| | |
|---|---|
| Humeri | Length 10.7 inches |
| | Separation from each other 1.5 inches |
| Diaphysis | Outer humerus 0.5 inches |
| | Inner humerus 0.75 inches |
| Humeral head | Outer humerus 1.5 inches |
| | Inner humerus 1.25 inches |
| Scapula | Medial length 9 inches (left was 4.5 inches) |
| | Upper border 2½ inches above 1st rib—6.5 inches |
| | Outer border 6.5 inches (left was 5.5 inches) |

Between the ulnar sides of the upper and lateral hands a web formation was noted. This ended at the level of the middle and distal phalanges. It contained in its distal border two stumps with bony spurs simulating distal phalanges of rudimentary fingers. At the level of the distal carpal row a solitary bone about 1.5 inches wide might be considered as an os centrale.

In summary, one may say that the upper hand, *A*, was dorsiflexed and pronated and the thumb was pushed under the other four fingers.

It articulated with a radius and ulna about 4 inches shorter than normal and had an incompletely developed carpal bone complex. The lower hand, *B*, most normal of the three, was volarflexed and supinated about 180 degrees against the opposite hand. It had a thumb in common with the lateral hand, *C*. The lateral hand was in 90 degree rotation opposite the other two and held midway between supination and prosupination.

## DISCUSSION

*Causes.* In searching for the origin of an abnormality such as the one here described, one must consider the possibility of endogenous or exogenous causes. The fact that the patient manifested no other abnormalities or defects and that none have been discovered in her family, apparently excludes an endogenous (hereditary) factor. Some type of damage originating from without during gestation is a more probable cause.

Various theories may give a greater or lesser approximation to the probable chain of events. Aschner-Engelmann[8] propound a gene or gene-complex which governs normal development; a pathologic opposing factor could, in their opinion, produce abnormal or supernumerary formations by affecting this gene. Non-specific injuries (pressure, temperature, chemical changes, etc.), by provoking diminished resistance of the genes, could thus possibly lead to abnormal development.

Dubrünner[9] points out that though the insult may be non-specific, the reaction of the cell is quite specific. The extent of variations produced would depend upon the development already achieved. The less differentiated the cell, the wider the variety possible. Greater progress in differentiation produces more resistance to a change in form.

Przibram[10] shows that disturbances of any nature may produce changes in *rate* of growth as well as in form. Trauma increases rapidity of growth until the injured part has once more caught up.

In our opinion, we can apply the Arndt-Schultz law to explain the occurrence of deformities, somewhat in the following manner: (1) minor stimuli cause irritation and result in overgrowth or division; (2) major stimuli cause inhibition and result in stunting, absence of division, webbing, etc., and, if exceptionally severe, will result in necrosis or loss of body parts.

According to the theory of Murk Jansen,[11] the degree of harm done depends on the rate of growth. Fast growing cells are especially vulnerable and may be affected by amniotic pressure. Excessive pressure may lead to ischemia, lack of nutrition and resultant growth disturbances.

To put all these theories together, we arrive at the following "explanation" for the abnormal development in the present case: Non-specific irritation may have occurred at a period of gestation when the right shoulder and arm were being formed but were not yet completely differentiated. Possibly an excessive amount of building material was present, and when the rate of growth was accelerated to make up for the harm done, a double humerus and plural hand resulted.

Some form of injury is thus the most probable explanation. What the irritating mechanism was we have no way of knowing, although the following may be considered:

1. Intra-abdominal pressure in pregnancy may have been abnormally increased, causing adhesions between the amnion and an extremity of the embryo. If expansion of the fetus and of the amnion did not take place in the same proportions, the points of adhesion might lead to superficial or deep grooves, and these might eventually become completely separated. In the case under discussion, grooves of separation appear down the arm, while the hands are separated completely.

2. Diseases of the amnion or of the uterine mucosa may adversely affect the embryo.[10] The fetus is well protected against injury, but inflammation or other disturbance of the maternal genital tract may have been present before conception. Insufficiency of amniotic fluid and excessive amniotic fluid have both been suggested as

causes of deformity. Doederlein suggests also that hemorrhage into the fetal membranes may be a factor. Usually, it is believed, the mechanism in these changes is pressure ischemia.

3. Physical or mental trauma, infectious and toxic influences, maternal genital pathology, etc., must also be considered. In certain cases, *x*-rays, particularly if applied in early pregnancy, have produced monsters. This may be excluded in our case.

4. Various experimenters have produced sports in animals by altering their milieu (or their bodily form). Loeb found that double monsters resulted from chemical changes in seawater; Hertwig produced spina bifida and other changes by alterations in temperature; Bodenstein caused duplication and reduplication of legs by interchanging the right and left legs in the embryos of butterflies or by turning the legs 90 degrees. Schade[12] produced deformities by calcium deficiency and changes in sodium chloride metabolism. Experimental fracture of an extremity in some cases resulted in duplication.

Whatever pathogenetic factor is postulated, in our case the spina bifida of the fifth cervical vertebra was presumably the result of an insufficient vertebral segment or a secondary injury to the area of the somites.

*Time of Injury.* It is interesting to speculate at what time the causative injury took place. Our conclusion is that it must have been very early in embryonic life, probably in the third or fourth week. At this time, two pairs of small, bud-like prominences or round tuberosities appear anteriorly at the sides of the embryo, representing the blastomatous arm mesenchyme. They later acquire a sort of mitten formation, without finger differentiation. The scapula is very high, at the level of the four lower cervical vertebral bodies. Injury or arrest of development at this stage would cause deformity of the whole scapula-arm-hand complex, since this period is one of rapid change and differentiation for the upper extremity. The diminished resistance of the extremity would make it unusually responsive to any noxa.

During the separation of the skeletogenous material, some factor produced segmentation, causing two humeri, two radii and three ulnae to materialize. A rotation produced an opposition of the third hand to the two others. The scapula probably separated originally and then fused into one giant scapula while the humerus, lower arm, and hand split in three different planes. As Klaussner pointed out, in duplication of the hand, the radial margins face each other with one thumb or none at all in the middle axis and suppression of the radial segment (note the missing radius of the lower hand). The upper hand shows so-called ulnar adaptation in a different plane.

Fracture of the scapula-arm-hand blastema in the third or fourth week may have caused a process of degeneration, followed perhaps by a process of super-regeneration with rearrangement of the skeleton. Perhaps as a result of this rearrangement, the additional ulna materialized and the olecranon was rotated in the ventral plane.

## SUMMARY

A case of congenital abnormality of the right arm and hand is presented in which the patient had duplication of the arm bones, terminating in a triple hand.

A survey of the literature fails to reveal a case like this, although duplication of extremities has often been reported.

An attempt is made to arrive at an embryologic explanation for the anomaly.

We may add that the patient feels in no way inhibited by her deformity (except for her shyness) and has absolutely refused any attempts at correction.

We are indebted to Dr. H. Kornfeld, of East Orange, for aid in the discussion of the embryology of this case.

## REFERENCES

1. Haller and Saviard. In Gould, G., and Pyle, W. L. Anomalies and Curiosities of Medicine, Philadelphia, 1898. W. B. Saunders.
2. Murray.

3. Bradford, E. H. Deformities and Paralyses of the Upper Extremity. 1917.
4. Joachimstal. Die angeborenen Verbildungen der oberen Extremitäten. Berlin, 1900.
5. Klaussner. Uber Missbildungen der menschlichen Gliedmassen und ihre Entstehungsweise. Berlin, 1900. Bermann.
6. Mueller, W. Die angeborenen Fehlbildungen der menschlichen Hand. Leipzig, 1937. Thieme.
7. Gruber, G. B. *Beitr. z. patb. Anat.*, 93: 1459, 1934.
8. Aschner-Engelmann. Konstitutionspathologie in der Orthopädie. Berlin, 1928. Springer.
9. Debrünner, Vom Wesen der angeborenen Missbildungen. *Arch. f. orthop.*, 34: 657.
10. Przibram, H. Teratologie und Teratogenese. Berlin, 1920. Springer.
11. Kieve, L. Zur Frage der Fruchtschädigung also Ursache angeborener Deformitäten und Krankheiten. *Ztscbr. f. ortbop. Cbir.*, vol. 59, 1936.
12. Schade, H. Zur endogenen Entstehung von Gliedmassen Defekten. *Ztscbr. f. Morpbol. u. Anthropol.*, 36: 375.
13. Bromann, I. Normale und abnormale Entwicklung des Menschen. Handbuch und Lehrbuch der Ontogenes und Teratologie. Wiesbaden, 1911.

Macrodactyly consists in a congenital overgrowth of one or more fingers or toes. The structures are perfectly normal in character, and merely gigantic in size for the age of the individual.

# THE ECTOPIC KIDNEY—A GYNECOLOGICAL DIAGNOSTIC PROBLEM*

## WITH CASE REPORT

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A PELVIC kidney is an infrequent, and, most often, an accidental finding in gynecological surgery. Because of its similarity to pelvic tumors, recognition is quite rare. When accompanied by gynecological symptoms the condition may lead to unwarranted laparotomy, and for this reason its preoperative identification is of utmost importance. When an ensuing hydronephrotic syndrome clinically suggests kidney displacement, intravenous roentgenography and retrograde pyelography are successfully employed for verification of the finding. In the case presented here, *x*-rays were taken, but, as will be seen, poor visualization of the affected kidney did not seem to warrant a further roentgen study, since the pain was not on the same side.

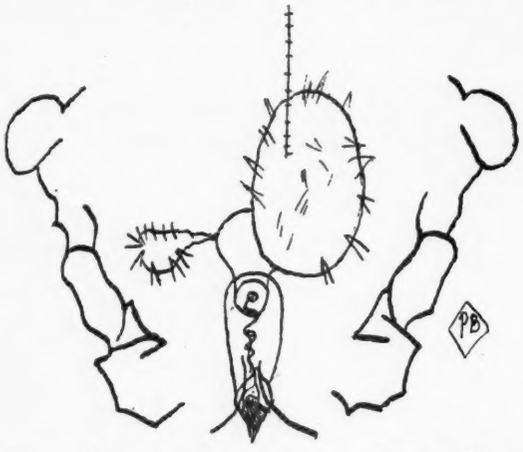


FIG. 1. Diagram of preoperative pelvic findings.

A brief presentation of this case is worthy of note because of the unusual features and atypical operative findings, which seem to clarify the cause of diagnostic error. The value of painstaking *x*-ray technique is demonstrated.

D. W., married, age 24 years, music teacher, entered Mt. Sinai Hospital on February 16, 1939. In childhood, she had had pneumonia and bronchitis. The latter recurred several times subsequently. In 1929 her sight was restored by operation after a three months' period of blindness. A left salpingectomy was performed four years before, for an unruptured ectopic gravidity.

The menses had always been normal (14 × 28 × 4). A six week amenorrhea since the last regular period on December 27, 1938, was followed by profuse, continuous bleeding, accompanied by severe, constant, low back pain and intermittent umbilical pain, which radiated into the medial and anterior aspects of the right thigh. There were also urinary frequency and urgency. Coitus without contraception occurred one week after the last period.

The patient, a short, thin, colored female appeared acutely ill, although the temperature was only 100°F. The pharynx was injected and moist râles were heard throughout the chest. The accommodation response of the pupils was sluggish. Pronounced midline abdominal tenderness was elicited. The left lower abdominal scar was firmly healed.

Upon pelvic examination, moderate uterine bleeding was observed. To the left was a tender, immovable, semi-cystic mass extending from the posterior pelvis to the anterior abdominal wall, to which it was fixed. In the right pelvis the indefinite contour of an adnexal mass could be palpated through thickened parametria. Examination under anesthesia was advised because of marked muscle rigidity.

*Laboratory Findings.* Blood pressure was 122/80. Sedimentation time was over one hour. Hemoglobin was 80 per cent; red blood cells 4,700,000; white blood cells 10,400; 66 per cent polymorphonuclear leucocytes; 30 per cent lymphocytes; 2 per cent eosinophiles; 1 per

* From the Gynecological Service of Dr. I. C. Rubin, Mt. Sinai Hospital, New York City.

cent mononuclear leucocytes. Urine, Wassermann and Aschheim-Zondek tests were entirely negative. Sputum examination revealed no tubercle bacilli. The chest *x*-ray was also negative.

Since the radiation of the pain suggested bladder or right renal tract involvement, an intravenous pyelography was done. Left pelvic concretions, probably phleboliths, were reported. The left kidney, however, was completely obscured by intestinal shadows. The right kidney was normal in size, shape and position. Inasmuch as there were no left renal signs or symptoms, rechecking the left kidney by *x*-ray was considered unnecessary.

Operation was delayed, however, until the cough subsided. The preoperative diagnosis under anesthesia was: left inflammatory cystic mass, adherent to the anterior abdominal wall underneath the abdominal scar; and also a right tender adnexal mass fixed in the anterior cul-de-sac. (Fig. 1.)

Eleven days after admission, under avertin and ethylene anesthesia, the abdomen was opened. The pelvic organs were hidden from view by adherent omentum, and the anterior abdominal peritoneum was firmly attached to the omentum overlying and attached to the large left retroperitoneal pelvic mass. (Fig. 2.) The entire right bladder vault was folded back proximally upon itself and fixed to the right adnexal mass by inseparable adhesions.

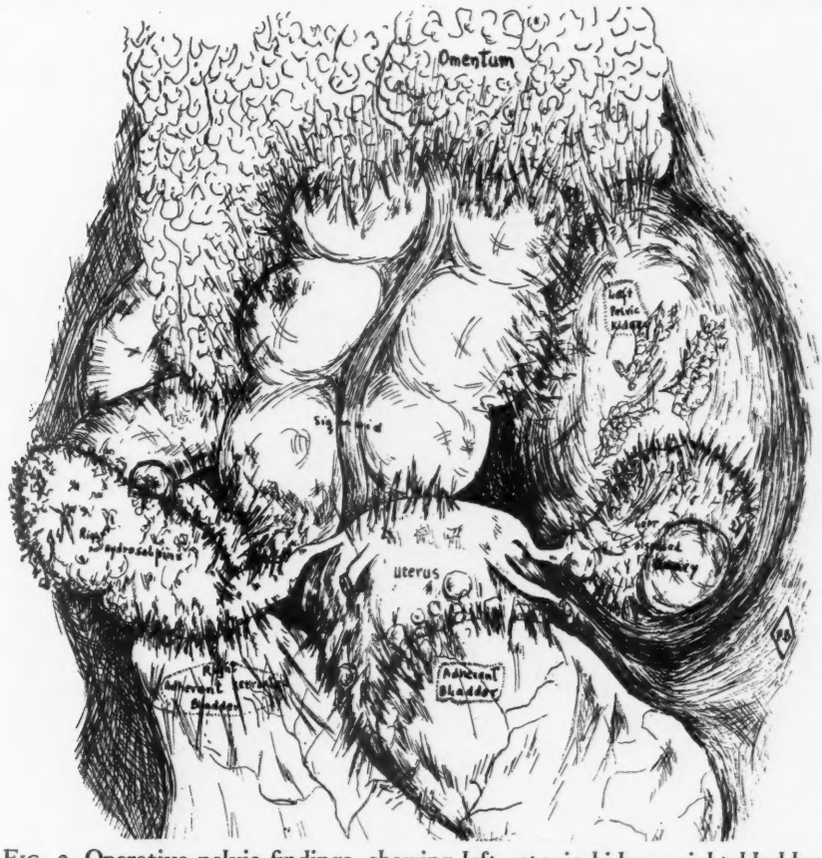


Fig. 2. Operative pelvic findings, showing left ectopic kidney, right bladder vault retracted by right adnexal adhesions and marked chronic pelvic adhesive peritonitis.

A small uterus was buried deeply beneath a cluster of seroceles and adhesions to the sigmoid and bladder. No vestige of the left tube was demonstrable. The left ovary, enlarged, cystic and buried, was dissected from adhesions surmounting the lower part of the above described retroperitoneal mass, which extended from the fourth lumbar vertebra into the true pelvis. The mass was firm and size of a grapefruit. It protruded forward and was adherent to the abdominal wall peritoneum underneath the scar.

Upon incising the thickened adhesions and the capsule, the left kidney cortex was observed. The capsule was immediately sutured.

On the right side a large retort-shaped tube and an enlarged diseased ovary were enucleated by blunt and sharp dissection from the anterior cul-de-sac and right bladder vault adhesions. was 11 cm. long. The wall was 5 mm. wide. The fimbriated end was closed, bulbous and retort-shaped, with surface adhesions. Proximally the lumen was obliterated.

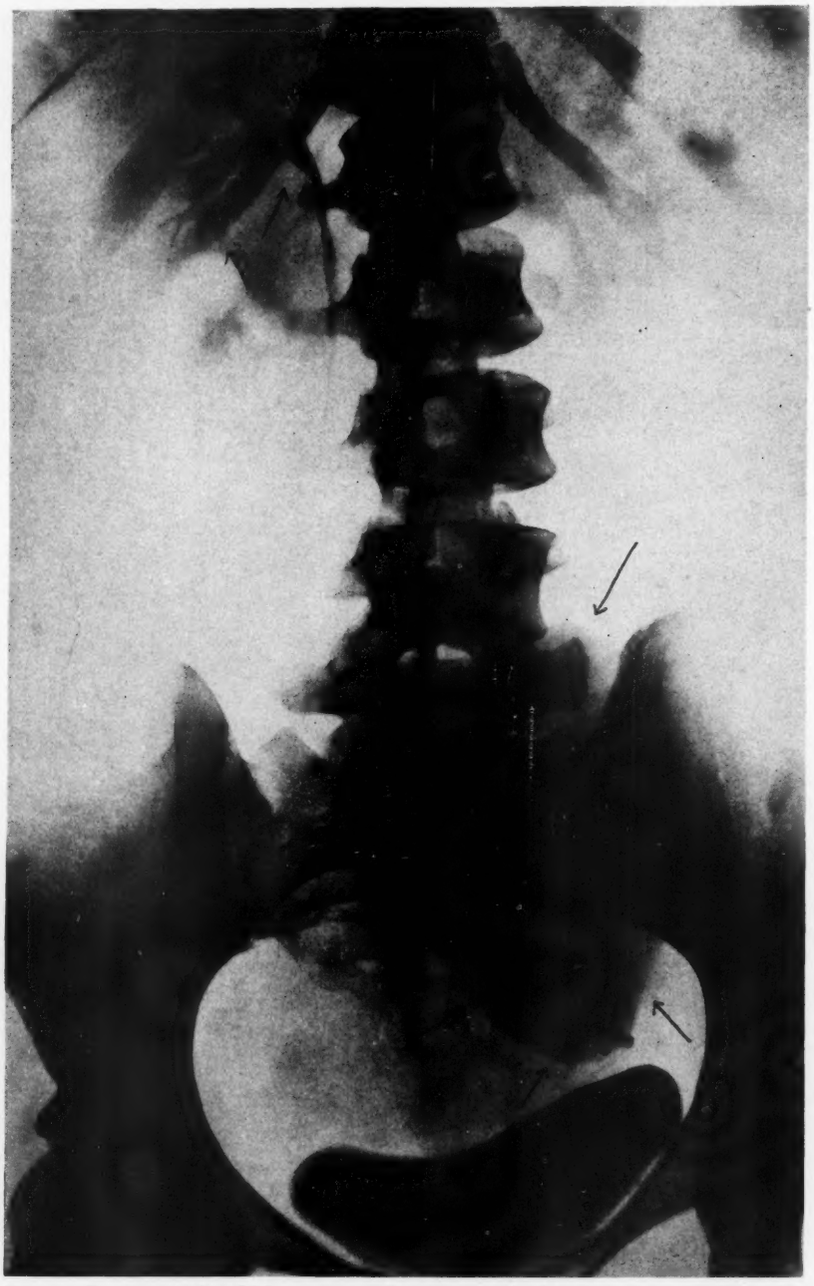

FIG. 3. Postoperative kidney *x*-ray (intravenous) showing clear outline of a left ectopic kidney.

The right tube and portions of both ovaries were removed, care being exercised to preserve the ovarian blood supply.

*Pathological report* revealed chronic salpingitis and hydrosalpinx; follicle cysts in diseased portion of ovaries. The Fallopian tube

*Postoperatively* there was right lower quadrant pain, tenderness, spasticity and dysuria. Right thigh pain persisted for ten days. There were also a productive cough and râles at both lung bases but these disappeared after eight days. During this time the temperature rose

to 102.8 degrees, but subsided within several days.

Two weeks after operation, March 16, 1939, a second *x*-ray (Fig. 3) of the urinary tract showed "left kidney in ectopic position in the presacral area."

The patient, observed at the follow-up clinic on May 12, 1939, ten weeks after operation, was free of symptoms. The left presacral mass was palpated very distinctly.

## SUMMARY

1. An interesting and undiagnosed case of asymptomatic ectopic kidney is presented. The error of omission committed here is of teaching value.

2. Contralateral bladder distortion from chronic inflammatory adhesions produced the urinary syndrome on the contralateral side.

3. Preoperative *x*-ray, because of intestinal shadows, did not reveal an ectopic kidney.

4. The importance of proper preparation of the patients for *x*-ray is stressed by this case. Postoperatively, pyelography confirmed the laparotomy findings of left ectopic kidney.

5. An ectopic kidney should be suspected when a low retroperitoneal pelvic mass is felt, even when it is fixed to the anterior abdominal wall.

6. The rarity of this condition, the atypism of the symptoms, and the instructive elements of the case warrant reporting.

# FALLACIES IN DIAGNOSIS OF RENAL AGENESIS

## WITH SPECIAL REFERENCE TO MICROSCOPICAL URETERAL ORIFICES

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THE greatest majority of reported cases of congenital solitary kidney were discovered at autopsy, the patient having died in most cases from causes unrelated to the lesion in the urinary tract. With the advent of the cystoscopic and intravenous pyelography era, the literature became replete with case reports in which a clinical diagnosis of renal agenesis was made. By far the largest series from a single clinic was reported by Braasch and Mericks[1] (sixty-nine cases). They were careful, however, to point out that the diagnosis was definitely established by autopsy or surgical exploration of the absent side only in twenty-seven cases and that in the other forty-two cases it was inferred from roentgenographic and cystoscopic data. A clinical diagnosis of renal agenesis was made in 131 cases of the total 696 cases* so far reported in the literature but only in forty-one instances, or 31 per cent, was the diagnosis corroborated either by autopsy (seventeen cases) or by operation (twenty-four cases). It is possible that in a number of unproved cases a rudimentary or a totally destroyed kidney really existed on the so-called absent side, which in some instances may have even communicated with the bladder by means of an orifice so minute that its presence could be detected only by microscopical sections.

The diagnosis of a solitary kidney ordinarily is based on the following findings:

1. The apparent absence of a ureteral orifice on one side on repeated cystoscopic examination in an otherwise normal looking bladder. In cases of advanced cystitis associated with thickened mucosa and with extensive edema this finding may not be of value since the orifice may be extremely difficult to locate. The possibility of an ectopic location of the ureteral orifice must also be considered, as the ureter may end, according to Eisendrath and Rolnick,[2] in the posterior urethra, the seminal vesicles, the vas deferens, or the ejaculatory duct in the male, and in the urethra, in one horn of a bicornuate uterus, in the wall of the cervix uteri, in the vagina, in the vestibule of the vagina, and in the persisting duct of Gaernter in the female.

2. Absence of indigo-carmine elimination on one side.

3. Absence of a kidney silhouette on plain roentgenogram.

4. Absence of elimination of opaque medium on one side in intravenous pyelography.

5. Roentgenographic evidence of renal enlargement and asymmetry of the psoas muscle shadows.

Neither one nor all of the above findings may be an absolute criterion of an absent kidney as may be gleaned from the following:

1. Failure to find a ureteral orifice in an otherwise normal looking bladder. The orifice may be so minute that its presence can be discovered only on microscopic sections. In the classical case reported by Wason,[3] the orificed was so minute as not to be visible to the naked eye and yet allowed urinary drainage. In her case the patient was an infant seven months old, apparently healthy until it was six months of age, when fever, diarrhea, and emaciation developed and death finally occurred. Autopsy showed advanced bilateral hydronephrosis and megaloureter. At the site of the ureteral orifices cystic projections were found which on most careful inspection showed no opening. A solution of thorium was then injected

* A copy of the complete bibliography on the subject and a chart upon which the above data is based will be gladly mailed by the authors upon request.

into both ureters but it could not be seen to escape through the protrusions. On standing for one hour, however, a small amount of the solution was found to have collected in the bladder. Serial microscopic sections were cut through the bulbous portions of the cyst in search of orifices which were found on both sides.

A somewhat similar case was reported by the senior author.[4] His patient was a boy 4 years of age who had a pyuria, intermittent attacks of pyrexia accompanied by chills, and pain in the bladder and both kidney regions. Since birth the child had been unable to pass urine in a free stream. Examination of the blood showed the presence of malarial parasites. The urine was loaded with pus. The phenosulphonephthalein output was only 10½ per cent in two hours; the blood chemistry was, however, normal. Cystoscopy showed a huge cystic swelling situated in the middle and on the left side of the bladder very close to the vesical neck. No orifice could be found on the left side. The intravenous pyelogram showed no elimination on either side. A diagnosis of a bladder cyst obstructing the urinary flow with advanced bilateral infected hydroureter-nephrosis was made. On suprapubic cystotomy a huge cyst was found on the left side of the bladder obstructing the vesical neck. This cyst did not contain a ureteral orifice but one was found situated to the left side of the cyst. The child died of uremia within twenty-four hours. Autopsy showed marked destruction of both kidneys, which were reduced to shells, and tremendously dilated ureters. There were two ureters on the left side, one of which ended, apparently blindly, in a huge cystic dilatation within the bladder. Careful scrutiny with magnifying lenses failed to disclose a ureteral orifice over the tumor, but microscopic serial sections cut through the lower end of the ureterocele definitely established the presence of an orifice which measured 7/40 cm. in diameter.

The two cases cited above indicate that an apparent absence of a ureteral orifice as determined by an unaided eye is not a criterion that such a condition actually exists as the ureteral opening may be demonstrated microscopically.

2. Chromocystoscopy. In the presence of a destroyed kidney, with or without a microscopic ureteral orifice, elimination of the dye will not take place.

3. A small atrophic kidney or a shell kidney may exist on the side where its absence is suspected and fail to cast a shadow on *x*-ray, or the *x*-ray may be so obscure that no renal silhouette can be made out at all.

4. The absence of dye elimination in intravenous pyelography may be due to a completely destroyed kidney.

Enlargement of the renal shadow, undoubtedly compensatory in nature, is a fairly uniform and a characteristic *x*-ray finding in cases of solitary kidney, as well as the increased size of the renal pelvis which occurs less frequently. Recently Braasch and Merricks[1] made an interesting observation that in 16 per cent of their cases the psoas muscle shadow was wider and that in 40 per cent there was an occurrence of less sharper definition of the psoas muscle on the side where the kidney was absent. These findings were noted in two out of the nine unpublished cases observed by one of us (R. L. D.) but cannot, of course, be construed as pathognomonic evidence of renal agenesis.

It can, therefore, readily be seen from the above that we really have not a single diagnostic criterion which determines clinically, with absolute certainty, that we are dealing with a congenital absence of one kidney.

The clinical significance of the foregoing lies in the fact that in a case in which a complete urologic survey discloses what appears to be a congenital solitary, but otherwise normal kidney, but which presents unexplained evidence of urinary infection, such as pyuria, pyrexia and pain on the side where the kidney is supposedly absent, an exploratory operation may be indicated to rule out the possible presence of a diseased, functionally dead kidney.

## CASE REPORTS

Case 1. A 20 year old colored female was admitted to the obstetrical service at Sydenham Hospital, December 12, 1936 with symptoms pointing to toxemia of pregnancy. She was apparently about seven months pregnant, gravida 1, para 0. For the last three months of her pregnancy she had had headaches and ankle edema. At the time of admission she appeared toxic, her blood pressure was 210 systolic, 130 diastolic, and ankle edema was present. Fetal heart sounds were not audible. The eyegrounds showed distended vessels.

The urine analysis disclosed 4 plus albumin, a few red blood cells and a moderate number of pus cells in clumps. The blood examination showed 74 per cent hemoglobin, 4,750,000 red blood cells, 14,300 white blood cells, 70 per cent polys, of which 16 per cent were immature, 16 per cent lymphocytes, and 4 per cent monocytes. The urea nitrogen was 56, uric acid 4.6, and glucose 79. The Van Slyke index was 77.9 (144 per cent of standard clearance).

On the night of admission the patient was delivered of a dead fetus, and improved rapidly thereafter. As her urine continued to be loaded with pus cells and red cells, she was referred to the urologic department for survey.

The plain roentgenogram (Fig. 1) showed a very dense but mottled, huge calcified area over the region corresponding to the position of the right kidney. The shadow extended from the eleventh rib to a level just below the transverse process of the second lumbar vertebra. There was no silhouette of a kidney shadow surrounding this calcified area. In view of subsequent findings which pointed to absence of kidney on same side, this shadow, especially because of its mottled appearance, could have easily been mistaken for one of extrarenal origin.

The intravenous pyelogram (Fig. 2) showed a complete absence of dye elimination on the right side. The left ureteropelvic tract was visualized very well and there was the characteristic compensatory dilatation of the pelvis and all of the calyces and ureter commonly observed in cases of solitary kidney.

Cystoscopy on January 3, 1937 disclosed no ureteral orifice on the right side, and on prolonged inspection indigo carmine, which was injected previously by the intravenous route, was not seen to emerge from any point on that side. On the left side the dye came through in powerful jets of excellent concentration. The urine obtained from the left kidney was clear and on microscopic examination showed no abnormal constituents. The bladder urine, although laden with pus and red blood cells, showed neither tubercle bacilli nor any other organisms.

Two days later the patient was cystoscoped by another member of the urologic staff with special reference to localization of an orifice on the right side. This time again no orifice could be found on very long and conscientious inspection. The bladder mucosa showed no evidence of any inflammatory lesion. It appeared perfectly smooth and normal throughout, and a ureteral orifice, if present, should therefore have been found with ease.

A tentative diagnosis was made of a tuberculous right kidney undergoing an autonephrectomy through calcification (putty kidney), having an atrophied ureter with a microscopic opening into the bladder. It was believed that the presence of a focus of infection was responsible for the toxemia of pregnancy and that removal of this focus would prevent a similar episode in the future.

An exploratory operation was performed by Dr. S. Malisoff on January 8. A small atrophied right kidney was found which was removed. The right ureter was explored with a probe which could be passed only for a distance of 2 inches. Methylene blue was then injected into the ureteral stump which was then ligated. *One-half hour after the operation urine obtained by catheterization of the bladder was faintly blue and two hours later a specimen had a marked blue color, which proved beyond any doubt that a communication existed between the right kidney and the bladder. This explained the presence of pus in the urine.*

The removed right kidney measured 7 by 4.5 by 2.5 cm., was markedly atrophied, and was covered by adherent perinephritic fat which had undergone fibrolipomatous changes. The capsule stripped with difficulty. On section the kidney was found to consist of many sacs, each filled with a gritty material which had undergone calcareous changes. The sacs also contained a great deal of cheesy material. No actual calculi were present. Unfortunately, after being photographed, the specimen was misplaced so that no microscopic section could be made. Grossly, however, the specimen was undoubtedly that of a tuberculous (putty) kidney.

The postoperative course of the patient was uneventful and she was discharged January 21, 1937.

FIG. 1. Note the huge mottled shadow in the region of the right kidney. Cystoscopy showed no ureteral orifice and no elimination of indigo-carmine on same side. The case could have readily been mistaken for a congenital solitary kidney and an extrarenal calcification on the right side.

FIG. 2. Same side. Intravenous pyelogram showed no elimination of dye on the right side. Note the uniform dilatation (compensatory) of the left ureteropelvic tract, characteristic of a solitary kidney.

Careful search of the literature revealed but one other case, reported by Uthoff[5] similar to the one cited above. In his case, although cystoscopy and intravenous pyelography pointed to the presence of a solitary kidney, an exploratory operation was nevertheless performed on the "absent" side which disclosed a large calcified cystic kidney mass. In his case a female patient complained suddenly of severe pain in the region of the "absent" kidney. As the urinalysis showed pus and blood and as the investigation of the bladder and the opposite kidney showed no evidence of any lesion, we would venture to suggest that in his case, as in our own, blood and pus found its way into the bladder from the destroyed kidney through a microscopical ureteral opening.

McKenzie and Hawthorne[6] emphasize that in 3 of their series of five cases of renal aplasia, pain was for unexplained reason complained of on the side of aplasia. It can be readily seen that the presence of a rudimentary, trouble-giving kidney, might have been entirely overlooked had the maldevelopment reached a slightly higher degree, resulting in the absence of a visible ureteral orifice. In a few of the reported cases of solitary kidney in which cystoscopy did not show a ureteral orifice on one side, pain was for some "unexplained reason" a prominent symptom on the side where the kidney was supposed to be absent.

CASE II. N. S., age 27, a white male, single, was admitted to Sydenham Hospital on June 26, 1936, complaining of high fever, nausea and pain in the pelvic region. There were no symptoms of bladder irritability. The patient denied having had gonorrheal infection but stated that occasionally he had received prostatic massage.

His temperature on admission was 106°F. which gradually returned to normal on July 19. During this time physical examination was entirely negative. The prostate gland as felt per rectum was normal and there was no evidence of urethritis. The *x*-ray of the urinary tract was unsatisfactory and that of the chest showed no lesion. The urine was laden with pus, blood and

bacteria. Blood cultures were negative at the end of twenty-four, forty-eight and seventy-two hours. The Widal was negative. Blood examination for melitensis and typhoid and paratyphoid A and B was likewise negative. The blood chemistry was normal.

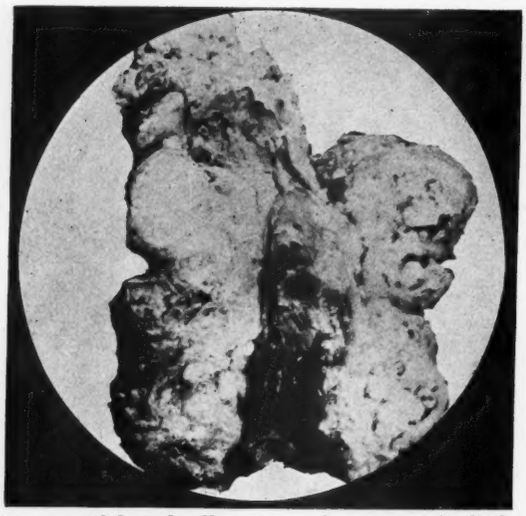

FIG. 3. Although all *x*-ray and cystoscopic findings pointed to absence of a kidney on the right side, an exploratory operation was performed because of unexplained pyuria. Cross section of atrophic tuberculous (putty) kidney removed at operation. The organ measured 7 × 4.5 × 2.7 cm.

On July 20, the patient began to complain of slight burning on urination and his temperature rose again to 102.4°F. and ran an irregular course, going up as high as 104.6°F. until July 31 when it dropped down to normal again.

Three cystoscopic investigations during this period failed to visualize a ureteral orifice on the left side; likewise there was no indigo carmine elimination on the same side. The output of the dye was excellent from the right orifice. The bladder wall was trabeculated and the mucosa slightly reddened. The urine obtained from the right kidney was clear and contained on microscopical examination but an occasional leukocyte.

Two intravenous pyelograms at twelve day interval failed to visualize the left ureteropelvic tract. The right tract showed a moderate dilation characteristic of a solitary kidney.

As every other condition was excluded with a fair degree of accuracy, it was evident that the patient had a urinary tract infection. He had neither the clinical manifestations nor the cystoscopic evidence of acute bladder infection or urethritis and the examination of the right kidney urine likewise could not explain the source of his heavy pyuria. We suggested that the patient might have an acute exacerbation of an old infection in a destroyed kidney on the left side and that pus and blood found its way to the bladder through a microscopical ureteral orifice. An exploratory operation on the so-called absent side was therefore advised, but the patient refused to submit to it and was discharged from the hospital on October 20, 1936.

## SUMMARY AND CONCLUSIONS

1. Review of the literature indicates that of 131 reported cases in which a clinical diagnosis of renal agenesis was made, 41 cases or only 31 per cent were confirmed by either exploratory operation or autopsy.

2. The most thorough urologic investigation which includes cystoscopy, chromocystoscopy, plain roentgenography, and intravenous pyelography, cannot completely rule out the presence of a destroyed infected kidney with microscopic orifice on the so-called "absent" side.

3. The ureteral orifice may be so minute that its presence may be demonstrated on microscopic sections only.

4. A case is reported in which complete urologic survey showed what appeared to be a solitary kidney. Because of unexplained pyuria, exploratory operation was done, revealing an autonephrectomized (putty) kidney on the opposite side. Methylene blue injected into the ureteral stump was recovered from bladder shortly after operation, which definitely proved that a communication existed between kidney and bladder, explaining the pyuria.

## REFERENCES

1. BRAASCH, W. A. and MERRICKS, I. W. Clinical and radiological data associated with congenital and acquired single kidney.
2. EISENDRATH and ROLNICK. Textbook of Urology. Philadelphia, 1928. Lippincott.
3. WASON, I. M. Report of case of congenital stenosis of both ureteral orifices. *J. Urol.*, 4: 123, 1920.
4. DOURMAŞHKIN, R. L. Blind ureterocele. *Urol. & Cut. Rev.*, 39: 783, 1935.
5. UTHOFF, CARL J. Diagnosis of a case of solitary kidney with accompanying vestigial cystic remnant of other kidney. *Urol. & Cutan. Rev.*, 40: 559, 1936.
6. MCKENZIE, D. W. and HAWTHORNE, A. B. Unilateral renal aplasia, *Surg., Gynec. & Obst.*, 46: 42, 1928.
7. GUTIERREZ, R. Surgical aspect of renal agenesis, *Arch. Surg.*, 27–686: 735, 1933.

# POLYPOSIS OF THE COLON

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The Clinic

HONOLULU, T. H.

GENERALIZED polyposis of the colon is not a new clinical entity, but the realization of the frequent association of this condition with carcinoma of the large intestine has progressively broadened the field of its interest. Articles in recent literature by Erdmann and Morris,[1] McKenney,[2] Rankin and Grimes,[3] Miller and Sweet,[4] Coffey[5] and others, not only emphasize the frequency of malignant degeneration in this condition but call attention to the frequent familial tendency of the disease.

It is generally acknowledged that the lesions are of the adenomatous type and the term "multiple adenomatosis" proposed by Lockhart-Mummery,[6] is a more descriptive terminology but "polyposis" seems to be the more popular expression.

Erdmann and Morris are responsible for the clinical classification of polyposis of the colon into two groups, namely: (1) the adult (acquired type), and (2) the adolescent, or congenital disseminated type. This classification, while not serving to differentiate morphologically the individual variations frequently encountered, does on the whole serve as a useful working basis.

The acquired type presents itself in adult life, usually as a few scattered polypi secondary to frank evidences of chronic inflammation. They are, not infrequently, the precursors of malignant degeneration.

The adolescent (or congenital disseminated type) manifests itself early in life by intermittent diarrhea, rectal bleeding, abdominal cramps, loss of weight and secondary anemia. This is the type in which the familial nature of the disease is evident. Rankin and Grimes point out that these tumors are widely distributed in the colon, frequently extending from anus to cecum, but that their appearance in the sigmoid and the rectosigmoid regions is approximately eight times more frequent than in any other portion of the colon. This, they point out, is particularly significant in the light of the similar occurrence and ratio of the distribution of carcinoma of the colon, and immediately suggests a relationship.

While the average age of the individuals in which the condition is recognized is around the second and third decade of life, patients as young as 2 years and as old as 70 have been reported.

The diagnosis of the condition is easily made, the simplest means being by the palpating index finger substantiated by inspection through the rectosigmoidoscope. A colon *x*-ray, in addition, helps determine the extent of the involvement. In other words, if an examination is made that is called for by the patient's presenting symptoms, there should be no excuse for failing to arrive at a proper diagnosis.

The treatment of polyposis of the colon is surgical and when a generalized involvement is encountered, complete removal of the colon is the only assurance against subsequent serious complications. Isolated polyps of the colon may be dealt with individually, either by excision or fulguration by way of the proctoscope or transabdominally, but *x*-ray studies of the colon should be made at the time to see if polyps higher up can be demonstrated. However, when negative evidence is obtained, one has the feeling that possibly their presence is being overlooked. Just recently, an abdominoperineal resection of the colon was done because of malignancy developing at the site of removal of a polyp by Dr. Straub of our Clinic, nine years previously. In the removed specimen of rectosigmoid there

were other polyps and there may be more proximally situated to the point of excision that will give further trouble.

FIG. 1. *X*-ray after a barium enema. The filling defects caused by the polyps are characteristic, showing the extent of the disease preoperatively.

More recently, cases have been reported in which the colon has been removed to the rectosigmoid region, the polyps in this area being removed by fulguration and this part of the gut later utilized for anastomosis to the ileum. A permanent ileostomy is certainly to be avoided if this can be accomplished with safety to the patient, but this hardly seemed feasible in the case which we are reporting.

When complete excision of the colon is to be done, a preliminary ileostomy is imperative, since this places the colon at rest and permits a considerable degree of recuperation of the structures before extirpation. The contents of the colon can be thoroughly cleaned out; many virulent bacteria are meanwhile destroyed, making peritonitis at subsequent operation less likely; the loss of blood diminishes and meanwhile the patient's depleted reserve is built up by transfusions; etc. If the rectum is to be preserved, an initial procedure of anastomosing the ileum to this portion of the gut may be considered, but most observers apparently prefer doing this as the terminal step.

In removing the colon, this may be done in one stage or several, depending upon how the patient reacts to the ordeal. The operation may be discontinued at any time that it seems desirable.

## CASE REPORT

The patient, a Hawaiian male, aged 20, entered Queen's Hospital on March, 23, 1939, under the care of Dr. Min Hin Li, who made the diagnosis. He had apparently been perfectly well until two months before admission to the hospital, when he developed diarrhea, abdominal cramps, dizziness and chills. The stools were watery and frequently contained gross blood. His appetite was poor and during this time he had lost 20 pounds in weight. General physical examination on admission was negative. Blood examinations at this time revealed 55 per cent hemoglobin, 4,390,000 red cells and a white blood count of 15,900, with polymorphonuclears 78 per cent, eosinophiles 1 per cent, monocytes 3 per cent, lymphocytes 9 per cent. The red cells showed many macrocytes, much poikilocytosis and considerable achromic staining. Repeated stool examinations, including cultures, showed nothing abnormal except for a strongly positive test for blood. The urine was negative, as were the Wassermann and Kahn tests. On March 25 and 30, transfusions of 500 cc. of citrated blood were given; the blood count on April 1 revealed hemoglobin 70 per cent, red blood count 4,780,000, white count 15,200, polymorphonuclears 61 per cent, eosinophiles 3 per cent, monocytes 4 per cent, and lymphocytes 32 per cent.

Digital and proctoscopic examination of the rectum revealed many polypi, but microscopic sections from biopsies of the most suspicious appearing ones showed nothing suggestive of malignancy. There was much oozing of blood from the irritated polyps during this examination. Colon *x*-rays indicated a generalized involvement of this structure by the polyps.

The temperature record previous to surgery showed only an occasional rise to 99°F.

No relative had apparently suffered from a similar condition. However, it is easily possible that this part of the history might be in error.

The patient left the hospital on April 6, feeling better, but was readmitted eight days later because of a recurrence of the symptoms for which he had originally come. Blood examination at this time showed hemoglobin 75 per cent, with 4,530,000 red cells and 25,450 white blood cells, with 75 per cent polymorphonuclears. Stool examinations continued uniformly positive for blood.

FIG. 2. Specimen removed at second operation (first operation ileostomy). Appendix at left, with its mucosa also polypoid; cecum and first part of colon. Note the long pedicle of the polyp above, and the size of the one below, backed by white paper.

FIG. 3. Specimen removed at third operation, including the second portion of the colon, the splenic flexure, which was not so heavily studded with polypi, and the first portion of the sigmoid.

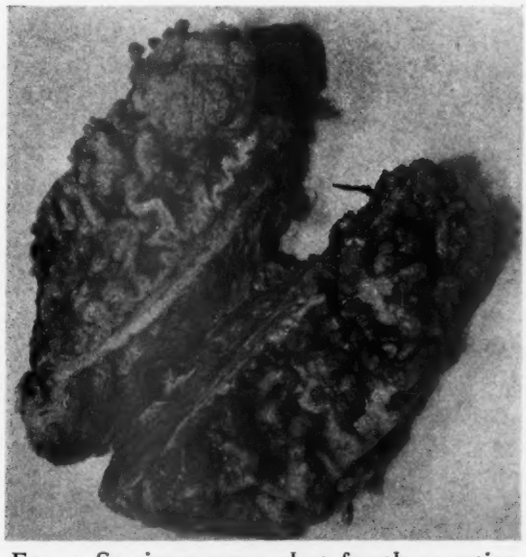

FIG. 4. Specimen removed at fourth operation, consisting of rectum and perirectal tissues, sliced longitudinally to show muscular and mucosal layers.

It was felt at this time that there was no treatment for this condition except surgery, and that a complete removal of the colon was necessary due to the large number of polypi visible and palpable in the rectum. The patient was transfused on April 19 and 24 and the first stage operation was done on April 25.

Under spinal anesthesia (pontocaine) a McBurney type of incision was made. The ileum was divided just proximal to its termination in the cecum, with the cautery between Stone aseptic intestinal clamps. The distal end was turned in and dropped back. The proximal end of the ileum was mobilized so that it could be drawn out beyond the abdominal wall for approximately 4 inches. This was accomplished by severing the mesentery near the root, first compressing the vessels before severing, to see if the circulation in the ileum to be mobilized remained adequate. It is a point worth remembering that only a small amount of mesentery can be divided near the bowel without interfering with the circulation, while a considerably greater amount can be divided near the mesenteric root without trouble, due to the anastomosing loops of vessels distal to this region.

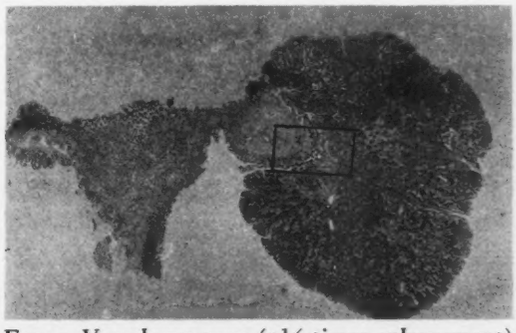

FIG. 5. Very low power (3½ times enlargement) of an entire polyp, showing, on the right its stretched base of relatively normal colon mucosa, on the left the structure of the polyp itself.

The proximal ileum was drawn well out of the wound, attached to the peritoneum and fascia, and the incision closed around it. A large sized Pezzer catheter was then placed into the pro-

truding ileum and the gut wall inverted around it. By this means the intestinal contents during its most irritating period, was conveyed away from the abdominal wall and at no time during the postoperative period was there excoriation of the skin. Tube drainage of the intestinal contents was successful for two or three weeks. After removal of the tubes the skin was protected by zinc oxide sprinkled with kaolin and overlaid with vaseline gauze.

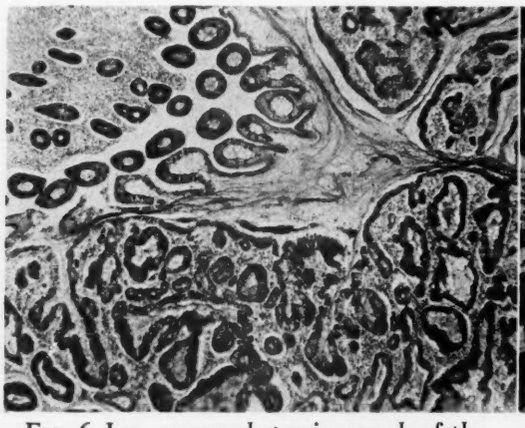

Fig. 6. Low power photomicrograph of the area outlined in Figure 5, showing normal structures at the right, below, and the adenomatous polypoid structures above and at left. Note the difference in the character of the glands, and the loose gelatinous type of the stroma of the polypoid portion.

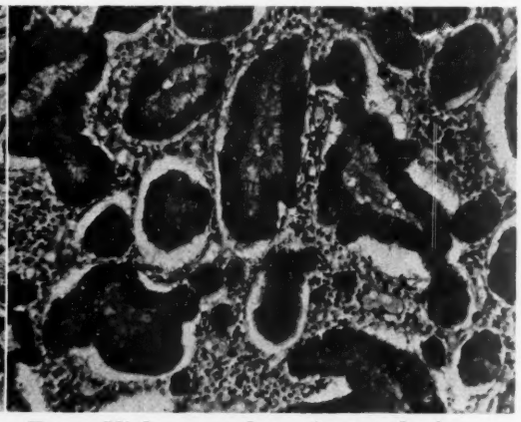

Fig. 7. High power photomicrograph of central portion of a polyp to show the mucoid type of the essential cell of the gland, and the loose, gelatinous type of the stroma.

Between the first and second operations, the patient was given another transfusion of 500 cc. of blood.

On May 12, or seventeen days after the first operation, again under spinal anesthesia, a long left rectus incision was made with a transverse incision to the right across the right rectus muscle between the umbilicus and the costal margin.

The cecum and ascending colon were mobilized by an incision along the lateral side, the vessels ligated, and the dissection carried over to the region of the splenic flexure. The omentum was freed from the transverse colon and preserved. Posteriorly, the ureter and duodenum were identified and protected. The dissection was rather difficult due to adhesions, edema, and the enormous size of the mesenteric glands. Examination, microscopically, of some of these glands revealed no malignant deposits. By the time the splenic flexure had been reached, the patient's condition did not justify prolongation of the operation. The incisions were closed up to the protruding colon, the Stone clamp was again applied, the gut cut off with a cautery and an aseptic closure of the gut lumen carried out, following which it was dropped back into the abdominal cavity and the incision closed. The patient was given 500 cc. of blood and intravenous saline and glucose during the operation.

On May 30, eighteen days following the second operation, a third operation was done under spinal anesthesia. A left rectus incision was made with transverse incision across the left rectus muscle in the splenic region. The remaining colon was removed into the hollow of the sacrum. The superior hemorrhoidal artery was preserved in order to prevent loss of viability to the upper end of the rectum. The distal end of the colon was inverted, dropped back and covered over by peritoneum from each side of the pelvis, forming a diaphragm of peritoneum between the upper and lower pelvis similar to that formed during an abdominoperineal resection for carcinoma of the rectum. The raw areas left by removal of the colon were closed over by sewing together the leaves of the colonic mesentery. Through an extraperitoneal stab wound in the left McBurney region, a rubber tissue drain was carried down to the stump of the distal colon and one carried upward below the left renal area.

Again, the patient was supported during operation by intravenous saline and glucose and by a blood transfusion.

Twenty-seven days after the third operation a fourth was carried out with the patient under spinal anesthesia, in prone position, with head

lowered, feet lowered, and legs separated. A longitudinal incision over the midportion of the lower sacrum was carried down and around the anus. The coccyx was removed. The rectum and anus were then extirpated without much difficulty, after ligating the middle hemorrhoidal vessels. The cavity remaining was packed with vaseline gauze and rubber tissue drains and the wound partially sutured around these drains. The patient was again transfused at the completion of the operation.

One month after the last operation, the patient was doing well, and the fecal discharge from the ileostomy was becoming more solid and less frequent.

REFERENCES

1. ERDMANN, J. F. and MORRIS, J. H. Polyposis of the colon. *Surg., Gynec. & Obst.*, 40: 460, 1925.
2. MCKENNY, D. C. Multiple polyposis of colon: familial factor and malignant tendency. *J. A. M. A.*, 107: 1871, 1936.
3. RANKIN, F. W. and GRIMES, A. E. Diffuse adenomatosis of colon. *J. A. M. A.*, 108: 711, 1937.
4. MILLER, R. H. and SWEET, R. H. Multiple polyposis of colon; a familial disease. *Ann. Surg.*, 105: 511, 1937.
5. COFFEY, R. J. Multiple adenomatosis of the colon; a clinicopathologic study. *Proc. Staff Meet., Mayo Clin.*, 13: 541, 1938.
6. LOCKHART-MUMMERY, J. P. The causation and treatment of multiple adenomatosis of the colon. *Ann. Surg.*, 99: 178, 1934.

THERE is ample proof that polyposis intestini is an inheritable disease, and Dukes has published charts and thirteen family pedigrees which are most convincing on this point.

THE brief excerpts in this issue have been taken from "Manual of Surgery" by Rose & Carless (Williams & Wilkins Company).

# HYPERPARATHYROIDISM

## REPORT OF A CASE OF PARATHYROID ADENOMA WITH CYSTIC DISEASE OF THE BONES

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THE interest of the orthopedic surgeon in diseases of the parathyroid glands is centered chiefly in the cases of hyperactivity of these organs causing a loss of the calcium content of the bones with resultant osteoporosis, deformity of the limbs and pathologic fractures. Stimulated by the work of Ballin of Detroit, the members of my staff at the Hospital for Joint Diseases and I have been carefully scrutinizing all bone lesions which come under our care for evidences of hyperparathyroidism. Cases of arthritis, localized and general osteoporosis and cystic disease of bones have been thoroughly studied, with particular reference to the serum calcium and phosphorus and the phosphatase. Our attention has been focused on the significance of these chemical changes through the work at our hospital of Dr. Henry L. Jaffe and his associates, whose studies are well known to many in the profession. Yet it was not until recently that there came under our care a patient exhibiting the characteristic manifestations of hyperparathyroidism arising from an adenoma of one parathyroid gland, the first case, I believe, in the records of our institution. The experiences of this patient so accurately mirror the changes in hyperparathyroidism and the effects of surgery that they are worth reporting in detail.

### CASE REPORT

The patient, a man of 49, had been known to me for fifteen years, but consulted me professionally for the first time in September 1935 for what appeared to be an idiopathic left sciatic neuralgia. This was readily relieved by simple physiotherapeutic measures. Certain observations were made then which did not seem very significant, but which in retrospect have manifestly an important bearing both on his present condition and perhaps, too, on the etiology of the sciatica. The history suggested that the patient's height had diminished somewhat during the preceding few years. A condition of stooped or round shoulders which had been present for many years was said to have become exaggerated. I found a moderate right dorsal idiopathic kyphoscoliosis known to have been in existence for many years. An increase of this deformity would reasonably explain the reduction in the height, the exact extent of which was uncertain. I noted also an enlargement of the head, but did not appreciate that this was pathologic, since I did not have a clear recollection of its previous size and shape. There were no neurologic disturbances. An *x*-ray film of the pelvis revealed an asymmetry which I considered as secondary to the scoliosis. The bones of the pelvis revealed no alteration in their architecture other than the morphologic change. Since the sciatica yielded to treatment, the man resumed his work as a printing compositor and I dismissed the case from my mind.

The patient returned three years later, on February 11, 1938, because of a fracture of his left arm which had occurred three months previously during a rather mild effort to go through a narrow doorway by pulling himself up with his left hand holding on to the framework of the door. More significant was the presence of symptoms of a severe systemic disturbance. He had grown progressively weaker so that he could walk only a few blocks at a time, and because of this, he had given up his work some time before the fracture incident. As his wife conducted a boarding house he contented himself doing odd jobs around the house. But willing as he was, he had little resistance, and even mild efforts tired him readily. He had lost some weight and several inches in height, his lower limbs had become bowed and his head was enlarged, noticeably so now to his wife and friends.

The examination showed a greatly changed individual. His stature was decidedly shortened; his skull was enlarged and by contrast his

face was small; his back presented a severe kyphoscoliosis with telescoping of the chest into the pelvis. The thighs were bowed outward. He walked slowly, awkwardly and with a waddling gait. His voice was weak. He appeared asthenic. In the left arm he had an ununited pathologic fracture of the shaft of the humerus. (Fig. 1.)

It was now, of course, apparent that we were dealing with a lesion that had produced a profound constitutional disturbance which was very marked in the osseous system. A parathyroid tumor was suspected and the patient was admitted to the Hospital for Joint Diseases for further study. Roentgen ray films were made of the entire skeleton. These showed very extensive involvement of many of the bones.

Table 1

BLOOD CHEMISTRY IN A CASE OF PARATHYROID ADENOMA

| | Calcium | Phosphorus | Phosphatase | Urea N. | N.P.N. | Sugar |
|---|---|---|---|---|---|---|
| Preoperative..... 2/14/39 | 13.0 | 2.7 | 23.1 | 31 | 54 | 92 |
| Operation..... 2/26/38 | | | | | | |
| Postoperative.... 2/28/38 | 8.4 | ... | .... | .. | 80 | 97 |
| 3/8/38..... | 5.4 | 2.8 | | | | |
| 3/11/38..... | 5.8 | | | | | |
| 3/14/38..... | 5.8 | 3.4 | | | | |
| 3/22/38..... | 5.2 | 4.2 | .... | .. | 32 | |
| 3/28/38..... | .... | ... | .... | .. | 31 | |
| 3/30/38..... | 5.0 | 5.1 | | | | |
| 4/6/38..... | .... | ... | .... | .. | 33 | |
| 4/8/38..... | 5.6 | 4.3 | | | | |
| 4/18/38..... | .... | ... | .... | .. | 34 | 97 |
| 4/21/38..... | 5.9 | 4.0 | | | | |
| 4/25/38..... | 6.2 | ... | .... | .. | 39 | |
| 5/2/38..... | .... | ... | .... | .. | 33 | |
| 5/3/38..... | 6.1 | 4.0 | | | | |
| 5/10/38..... | 7.0 | 3.8 | .... | .. | 34 | |
| 5/16/38..... | 8.0 | 3.4 | .... | .. | 34 | 101 |
| 7/28/38..... | 8.3 | 3.9 | | | | |
| 3/3/39..... | 10.2 | 2.9 | 4.4 | | | |

*X*-ray examination of the skull disclosed extreme thinning of the tables of the bones with complete loss of the trabecular markings (Fig. 2) but no definite tumor formations. There was extreme atrophy of the inferior maxilla with a large cyst in the symphysis. An extreme kyphoscoliosis of the dorsal spine was accompanied by pronounced wedging of all the vertebrae, associated with marginal hypertrophic changes. The ribs showed marked osteoporosis. In the scapulae there was almost complete loss of all bone detail due to resorptive changes. The lumbar spine also disclosed extreme generalized osteoporosis. No compression fractures were noted and the kidney showed no calculi. In the pelvis there was extreme generalized osteoporosis without tumor formation.

The left humerus (Fig. 1) showed evidence of a pathologic fracture through the middle third of the shaft and a large medullary tumor in the

Fig. 1. Pathologic fracture of left humerus. Note cystic condition at site of fracture and lack of callus. There is marked demineralization. Cortex is very thin.

same area. The alignment of the fragments was good but no callus formation was present. In the proximal extremity of the humerus extreme cortical thinning with granular disintegration was noted. The right humerus had a patchy area of osteoporosis at the junction of the proximal and middle thirds of the shaft, associated with moderate cortical thinning. The bones of both hands (Fig. 3) exhibited extreme osteoporosis with marked cortical thinning, presenting likewise the granular disintegration noted in the left humerus. Concentric atrophy was present in the shafts of the intermediate phalanges of the second, third, fourth and fifth fingers on the right side as well as of the shaft of the proximal phalanx of the fifth finger. Extreme cortical thinning of the right radius and ulna was more marked in the distal extremity of the ulna.

A large medullary tumor was present in the proximal third of the shaft of the right femur and in the proximal and middle thirds of the shaft of the left femur. Generalized osteoporo-

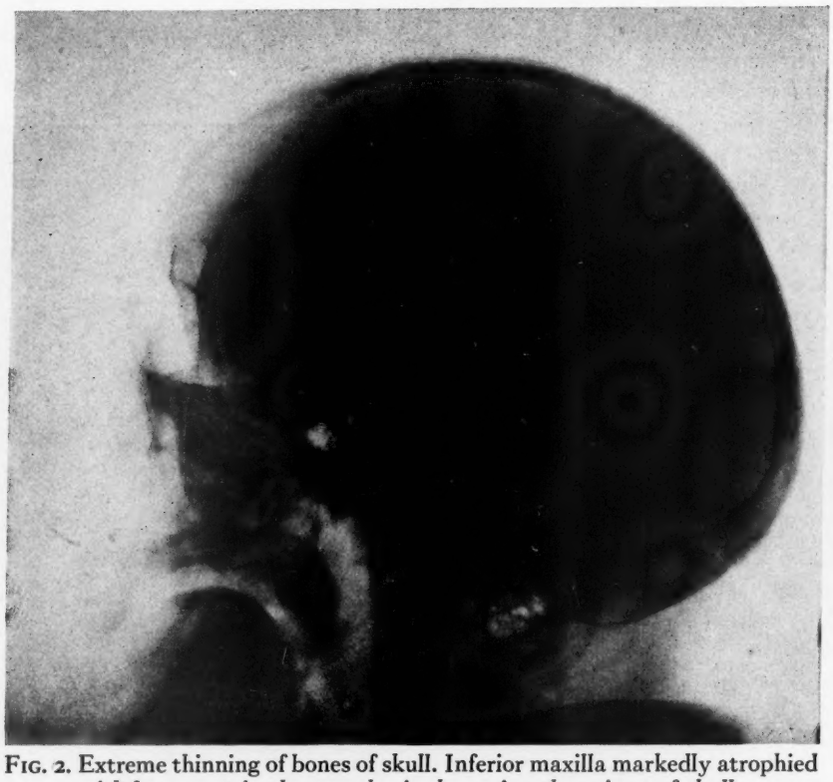

FIG. 2. Extreme thinning of bones of skull. Inferior maxilla markedly atrophied with large cyst in the symphysis shown in other views of skull.

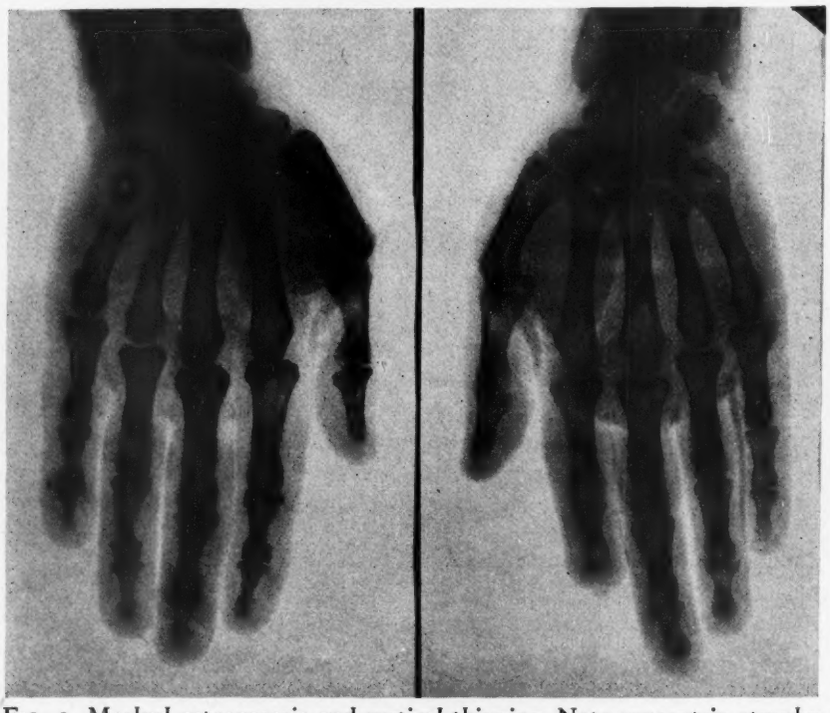

FIG. 3. Marked osteoporosis and cortical thinning. Note concentric atrophy in middle phalanges.

sis, but without the extreme cortical thinning, was seen in these bones also. Another large medullary tumor appeared at the junction of the middle and distal thirds of the shaft of the tibia (Fig. 4) while the anterior cortex of the tibia had been practically completely resorbed. In the right tibia a tumor had developed at the proximal third of the shaft. The bones of both feet disclosed marked osteoporosis without any evidence of tumor formation.

Fig. 4. Cystic area in shaft of left tibia. Marked resorption of anterior cortex.

Studies of the blood showed that the serum calcium (Table 1) was high and the phosphorus low, while the phosphatase was very high. There was in addition, a moderate secondary anemia. The sedimentation rate was 50. The Wassermann and Kahn tests were negative.

There seemed no doubt that we were dealing with a case of hyperparathyroidism (Drs. Jaffe, Bodansky and Pomeranz concurred). Dr. Milton Bodenheimer of the general surgical staff was invited to examine the patient. He thought that he felt an unusual mass in relation to the left lobe of the thyroid. The patient stated that he felt a mass in his neck moving during swallowing. However, other consultants were not certain of the presence of any tumor. Dr. Bodenheimer operated on this patient under general narcosis on February 26, 1938. He found and removed a tumor of the left inferior parathyroid gland, of the size of a small lemon. The other parathyroids were found to be normal in size, number, location and appearance.

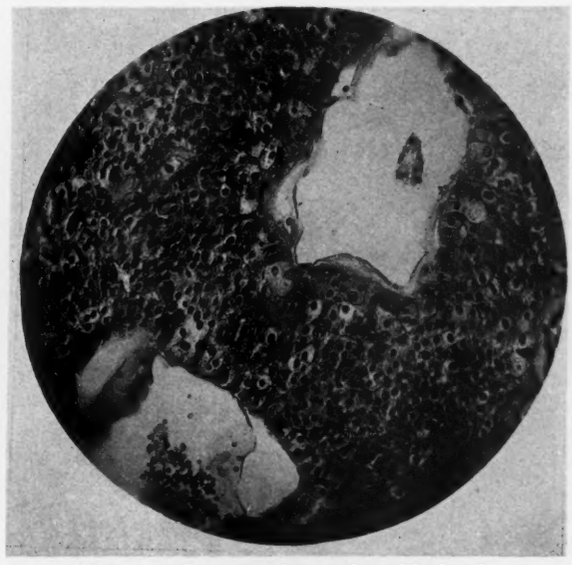

Fig. 5. Adenoma of parathyroid gland.

The tumor measured approximately 3.5 cm. in diameter and weighed 13 Gm. It was encapsulated and on section appeared very cellular. The cut surface had a grayish-yellow glistening appearance with small, irregular areas slightly more translucent.

Beneath the covering of dense fibrous tissue, the neoplasm was composed for the most part of a diffuse proliferation of moderately large polyhedral cells. The nuclei were moderately large, round, possessed a nuclear membrane, and showed a definite chromatin network. Others were more hyperchromatic and tended to be solid. An occasional nucleus showed a nucleolus. No mitotic figures were seen. The cells possessed abundant cytoplasm, granular and slightly eosinophilic. Water-clear cells were present but only near several areas of degeneration. The tumor contained numerous fine vascular channels and fibrous septa, but no attempt at gland formation was noted.

The pathologic diagnosis was chief cell adenoma of the parathyroid gland.

The patient was somewhat depressed for several days after the operation, but at no time seemed dangerously ill. Directly after the operation he received an intravenous injection of saline solution containing calcium gluconate and glucose, and thereafter he received calcium lactate and viosterol daily. The serum calcium dropped rapidly after the operation to 8.4 and

continued to drop more slowly so that at the end of a month, on March 30, 1938, it was 5 mg. per 100 c.c. of blood. The phosphorus in the meantime rose from 2.7 to 5.1.

FIG. 6. Postoperative *x*-ray fracture of left humerus healed. Cortex thickened. Porosis of bone diminished.

Clinically this patient was doing well and the operative wound healed by primary union. Hoarseness was noted on the day after operation, due to paralysis of the left vocal cord, evidently from involvement of the left recurrent laryngeal nerve. However, this vocal difficulty ultimately completely disappeared.

By March 9, 1938, eleven days after the operation, the patient was well enough to get out of bed. All during this time he had a positive Chvostek sign and at times showed restlessness and twitching of the eyelids, suggestive of latent tetany. On the night of March 11, thirteen days after the operation, his temperature suddenly rose to 103.5, he became very restless and noisy and had some difficulty in breathing, and became violent in his muscular activities. He was promptly given calcium gluconate intravenously and parathormone intramuscularly. Thereafter he received parathormone and calcium lactate daily until April 1, 1938. He improved a great deal and his temperature receded to normal.

Since April 1, 1938 the patient has continued to take calcium lactate and viosterol. His blood calcium gradually rose so that by May 16, 1938 it was 8.0; phosphorus was 3.4 and phosphatase fell from 23 to 4.0.

On May 20, 1938 the patient's general condition was good and his strength increasing. Firm union of the fractured humerus had occurred and *x*-ray plates showed an increase in bone deposits, especially in the long bones.

A year after the operation the patient was greatly changed: he was alert and ambitious, walked briskly and had acquired considerable physical endurance as compared with his former listless, weak state. A new set of *x*-ray films showed complete healing of the left humerus (Fig. 6) and a reduction of the cystic areas. The bones still looked atrophied and "washed out," but much less so than a year before. The general haziness was diminished and there was much more definite differentiation between the medulla and the cortex in all the long bones. The cyst in the shaft of the left tibia had almost completely disappeared. Similarly the cyst in the left femur was greatly reduced and the cortex of this bone had increased in density. The blood chemistry was almost normal. On March 3, 1939 the serum calcium was 10.2, the phosphorus 2.9 and the phosphatase 4.4.

## DISCUSSION

The original clinical picture, characteristic of hyperparathyroidism, included progressive weakness, enlargement of the skull, deformity of the spine and the lower limbs and a pathologic fracture of the left humerus. The diagnosis was confirmed by roentgenograms which showed a generalized demineralization and osteoporosis with an occasional cyst-like formation, and by the blood chemistry changes, marked hypercalcemia, hypophosphatemia and increase in phosphatase.

The postoperative reaction in this case was a hypoparathyroidism and tetany, including a marked drop in the blood calcium, a rise in phosphorus, restlessness, hyperactive reflexes and, finally, about two weeks after the operation, convulsions and a rise in temperature. The loss of parathyroid secretion was compensated by the administration of parathormone, calcium gluconate or lactate and viosterol.

The patient is now well on the way to a complete cure.

# BILATERAL COMPLETE SYNDACTYLISM OF ALL FINGERS*

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SYNDACTYLISM of the fingers of one or both hands of various degrees is a frequent congenital deformity. Complete involvement of all the fingers of both hands is, however, most unusual.

FIG. 1. Mother's hands. One finger has been removed with part of the corresponding metacarpal.

FIG. 2. Hands of younger sister of patient showing complete syndactylism of all fingers.

## CASE REPORT

A male infant was brought to the hospital at the age of 1½ years with a deformity of both hands existing from birth. The mother, age 21 had also had a deformity of both hands at birth. A photograph taken when she was 1½ years of age showed syndactylism of the fingers on both hands, but the exact degree could not be definitely made out. She had then been operated on and five fingers constructed on one hand and four on the other. (Fig. 1.) Scars of the plastic operations were visible and, although there remained some webbing, she had fairly good function of the fingers. Roentgenograms of her hands showed six metacarpals present on both the right and left hands, the extra one having been partially resected with the removal of the corresponding finger.

A recently born sister of the patient has complete syndactylism of all the fingers of both hands.† (Fig. 2.)

The patient had no abnormality of the head or of the special senses. Chest, abdomen, and spine were normal. The right and left hands

† Since submitting this paper similar operations have been performed on the sister of the patient restoring five fingers on both hands. (Figs. 8, 9 and 10.)

* From the Shriner's Hospital for Crippled Children.

were about the same in general appearance. (Fig. 3.) The fingers were joined together with skin extending up to the finger nails. Uniform flexion of the fingers gave the hands a cup-shaped form. (Fig. 4.) There were at least eight finger nails, some of which were fused together. Several of the extra fingers were quite rudimentary. Motion was limited, representing a combined movement of all the fingers. On roentgenographic study a conglomerate mass of phalanges was seen in various shapes and sizes, with six metacarpals on both hands. (Fig. 5.)

Fig. 3.

Fig. 4.

Figs. 3 and 4. Wayne, Mc. Patient before operation showing complete syndactylism of all fingers of both hands.

Fig. 5. Wayne, Mc. Roentgenogram showing six metacarpals with multiple extra phalanges.

The treatment consisted of a series of six plastic operations over a period of nine months from August 3, 1938, to May 3, 1939. Sufficient time was allowed between the operations to allow the infant to recuperate and also to attain the full effect of each operative procedure.

The following operations were performed:

1. Right hand. Three clefts were formed by cutting down between four sets of phalanges. The defects were covered with split skin grafts from the abdomen.

2. Left hand. Two clefts were formed by cutting down between three sets of phalanges.

Fig. 6. Wayne, Mc. Fingers after operation upon patient shown in Figure 3. Six fingers on each hand, one of which is to be sacrificed later.

Fig. 7. Wayne, Mc. Roentgenogram of hands after operation.

Fig. 8. Vesta McM. Palmar view of hands after operation showing five fairly well formed digits.

3. Right hand. Modified Didot operation further to separate the three units. Removal of accessory fingers, rudimentary finger.

6. Right hand. Plastic operation between thumb and adjoining finger. Deepening of cleft between two other fingers.

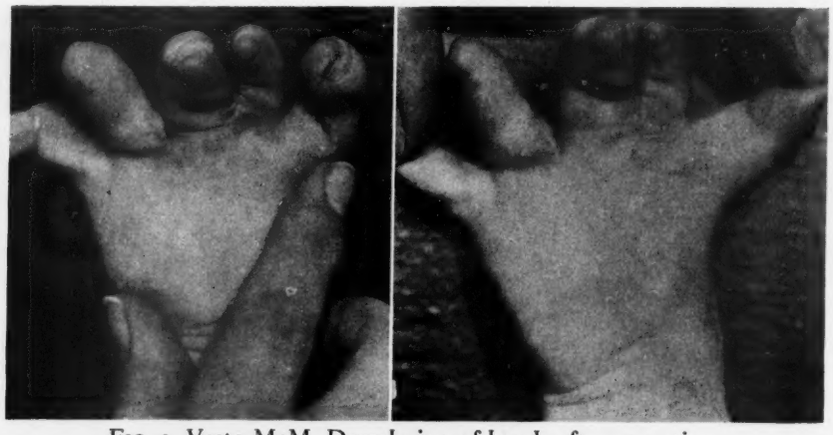

FIG. 9. Vesta McM. Dorsal view of hands after operation.

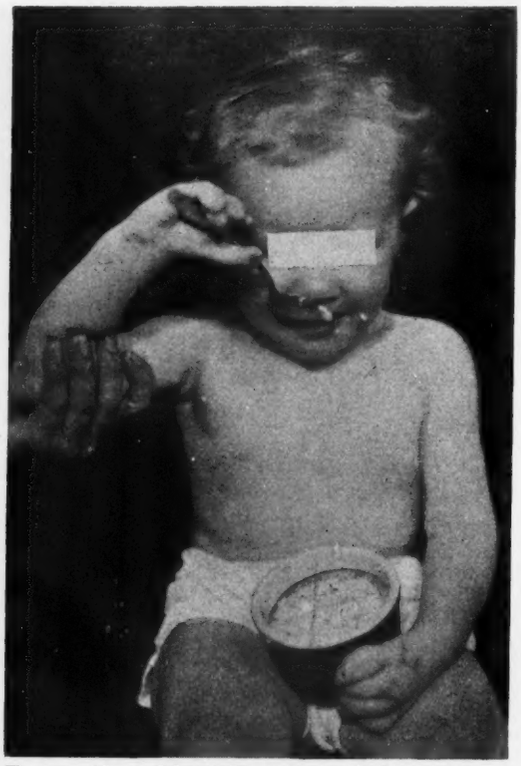

FIG. 10. Vesta McM. Patient feeding herself with one hand.

4. Left hand. Didot operation between middle set of fingers.

5. Left hand. Separation of two remaining sets of fingers.

Six fingers were formed on each hand. (Figs. 6 and 7.) The function of the fingers, considering the short time since operation, has been quite good. The patient can use the fingers of either hand for picking up small objects and for doing many useful things. It was thought advisable to allow the six fingers to remain for about a year in order to determine which was the least useful. At a later date it is contemplated to sacrifice one finger and perform whatever additional plastic work will be necessary.

## SUMMARY

1. Two cases of complete bilateral syndactylism were found in brother and sister. The mother of the patient had had a severe syndactylism of both hands. These deformities are of congenital origin and represent a regressive atavistic trait.

2. Restoration of six fingers with their corresponding metacarpals was secured on both hands in the one patient reported in this paper. He is able to do many things with either hand that he could not do before the operation.

# ACUTE CARDIAC COMPRESSION RESULTING FROM THE RESTRAINING INFLUENCE OF THE PERICARDIUM DURING ACUTE DILATATION OF THE HEART*

## OBSERVATION OF THREE CASES DURING PERICARDIAL EXPLORATION

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FROM first appearance, the title of this paper seems paradoxical—that the heart should be compressed by dilatation. However, such apparently is the case when dilatation is inhibited by the constricting influence of a pericardial sac that has reached its limit of elasticity.

In his discussion of the cardiac compression triads, Beck[1] lists intrapericardial hemorrhage as the most important cause of the acute cardiac compression syndrome. Hemorrhage may be the result of trauma, rupture of a myocardial infarct, a leaking aneurysm, and hemorrhage from neoplasms, scurvy, purpura and tuberculosis. Acute cardiac compression may be caused also by collections of exudates, transudates, and gas in the pericardial sac.

It is the purpose of this paper to present yet another possible cause of the acute cardiac compression syndrome based on the observation of three cases, two of which were operated upon by the author, and a third at which he assisted. This cause is the restraining effect exerted by the pericardium during acute dilatation of the heart.

Numerous series of gunshot and stab wounds have been reported from the larger charity institutions, especially throughout the south. Among these reports wounds of the heart have not been uncommon. Elkin[2] and Bigger[3] have recently summarized the diagnosis and treatment of these extreme surgical emergencies.

The surgical service of the University of Louisville and the Louisville City Hospital have treated a considerable series of heart wounds (Cox,[4] Mayer,[5] Griswold and Drissen[6]). The idea that the pericardium may be responsible for cardiac tamponade during acute cardiac dilatation, first presented itself during an operation carried out by the author at the Louisville City Hospital, on a patient with a stab wound of the chest.

CASE 1. W. S., a colored male, 40 years of age, was admitted February 20, 1938, thirty minutes after having been stabbed in the chest during a drunken brawl. He was orthopneic and complained of pain in the left shoulder and beneath the left scapula at the level of the fifth dorsal spine. (This physical finding of referred pain to the left shoulder and beneath the left scapula is interesting in that the same distribution of pain occurred in three other cases coming to autopsy, in which leakage of aortic aneurysms into the pericardial sac had produced cardiac tamponade.[7]) He presented a picture of severe apprehension and fear of impending catastrophe. His temperature was 97, pulse 70, respiration 30 and blood pressure 82/50. There was a minimum of external bleeding. There was a penetrating wound 1 cm. long in the third interspace, 3 cm. to the left of the sternum. The lungs were clear to auscultation and percussion. Cardiac dullness appeared to be within normal limits. Heart sounds were distant and barely audible, and there was distention of the neck veins. During examination the patient suddenly collapsed and became comatose and cyanotic. The blood pressure could not be obtained. He was taken to the operating room immediately and intravenous fluids started.

Under positive pressure cyclopropane-oxygen anesthesia, the chest was opened through a left parasternal incision with the horizontal limb of the incision extending laterally just below the nipple. The pectoral muscles were

* From the Department of Surgery, University of Louisville, and Louisville City Hospital.

reflected from the chest wall and the second, third, and fourth costal cartilages with the tips of the adjacent ribs, resected. The knife blade had entered the thoracic cavity 2 cm. lateral to the sternum. There was no blood in the pleural cavity and no injury to the pericardium could be seen from our exposure. The pericardial sac was very tense and appeared to be distended. In order to establish definitely whether the heart had been injured, it was decided to open the pericardium. While the pericardium was being split from base to apex, the heart bulged into the operating field as though it had been released from a tightly constricting covering. The normal amount of clear serous fluid escaped. Immediately the blood pressure became perceptible and rose to 90/50. During the next ten minutes of observation the circulation continued to improve and the heart returned to normal size. The pericardial edges were easily approximated with interrupted silk sutures. A 3 cm. opening in the one end of the pericardial incision was left for drainage into the pleural cavity. This method of draining the pericardium into the pleura suggested by Griswold,[8] insures against the recurrence of tamponade from excessive postoperative effusion.

The patient returned to the ward in good condition with a blood pressure of 80/50. During the next four hours the pressure gradually increased to 110/88 and became stable at that level. No additional intravenous fluids were given for fear of throwing additional burden on the heart. Convalescence was uneventful and the patient was discharged with a pressure of 125/95.

Shortly after Case 1, a second opportunity to observe the same phenomenon occurred. This time the unfortunate patient served unwittingly as a subject for the artificial production of acute coronary occlusion.

Case II. M. L., a colored female 20 years of age, was admitted March 8, 1938, about twenty minutes after having been stabbed in the chest with a pocket knife. Her temperature was 96, pulse 60, respiration 30, and blood pressure imperceptible. She was in profound shock, gasping for breath and did not respond to interrogation. Her breath smelled of liquor. The knife blade had entered the left chest in the fourth interspace, 2 cm. from the sternum. External hemorrhage, as evidenced by blood on the clothing, was moderate. Heart sounds were feeble and distant, rhythm regular, neck veins distended and cardiac dullness increased. There were no physical signs of pneumothorax or hemothorax.

The patient was taken to the operating room without delay and intravenous fluids started. Under positive pressure cyclopropane-oxygen anesthesia, the heart was exposed through a left parasternal incision, with resection of the third, fourth and fifth costal cartilages and the adjacent rib tips. The left pleura contained no blood. A laceration was found in the pericardium immediately beneath the external wound. On incising the pericardium from base to apex, the hemorrhagic tamponade was released and the heart resumed rapid activity, resulting in a terrific hemorrhage. The laceration in the heart was 6 mm. long, and had perforated the right ventricle immediately adjacent to and parallel to the right border of the descending ramus of the left coronary artery, in its proximal third. Hemorrhage was controlled by silk sutures deep in the ventricular wall, parallel to the laceration on either side. Free ends of the sutures were twisted to form a single strand on each side of the wound and then tied over the wound as a single suture. Several such sutures were required to control the hemorrhage. Although the coronary artery was not encircled by the sutures, it must have been occluded by the inverting effect. On control of hemorrhage from the heart, the blood pressure immediately rose to 120/80 and became stable. The heart, observed for fifteen minutes, became progressively cyanotic most markedly at the apex. To decrease the possibility of cardiac arhythmia as a result of handling the heart, 2 per cent novocaine solution was dropped over the surface of the heart early in the operation. The value of this procedure has been worked out experimentally by Mautz.[9] The cardiac rhythm remained regular and the blood pressure elevated, so it was hoped that the heart muscle had enough circulation to maintain activity. The pericardial edges were approximated without difficulty, using interrupted silk sutures, leaving a decompression opening into the pleural cavity at one end of the incision. The chest wall was closed in layers. As the skin sutures were being applied, the blood pressure suddenly disappeared. This was approximately twenty-five minutes after occlusion of the coronary artery.

It was feared that the coronary obstruction was responsible for what had happened, but to make certain that the heart wound was not leaking, the chest and pericardium were reopened. On release of the sutures in the pericardium, the heart fairly popped from the pericardial sac under great tension. The blood pressure instantly jumped to 160/120. The heart had become enormously dilated and bulged through the window in the chest wall. This presented another serious problem as the heart now began to lacerate itself against the ragged rib ends before it could be protected. This resulting new hemorrhage was as troublesome to control as the original heart wound. The entire heart was now extremely cyanotic. Edges of the pericardial sac were separated by 5 cm. and by no amount of tension on clamps fastened to the edges of the pericardium could the sac be pulled over the heart. Approximately forty-five minutes after the coronary artery had been occluded the heart stopped suddenly in diastole. Cessation of activity was preceded by no irregularities. After the heart stopped beating it could be compressed into a mushy mass, similar to that seen in toxic hearts at the autopsy table.

In retrospect, we feel that it is possible that this patient's life might have been saved had deep mattress sutures been used to close the heart wound, thus leaving the coronary vessels unobstructed on the surface of the heart. Some of these important technicalities, unfortunately, are overlooked when one is working under pressure, and on such an elusive object as a rapidly beating heart.

The next case had been reported previously by Mayer[5] in a series of cases from the Louisville City Hospital. It is the author's opinion that the case warrants reinterpretation in view of the above observations, and with Mayer's permission, it is re-presented here.

Case III. H. T., a colored female 43 years of age, was admitted September 7, 1934, a short time after having been stabbed over the pericardium with a pocket knife. She had been drinking heavily and was irresponsive. Her temperature was 98, pulse 58, respiration 20, and blood pressure imperceptible. The patient was very restless and apprehensive. In the fourth interspace, 6.5 cm. to the left of the sternal border, was a penetrating stab wound 1 cm. long. The clothing revealed little evidence of external blood loss. Physical signs showed an increase in cardiac dullness, but no evidence of pneumothorax or hemothorax. Heart sounds were regular but distant and weak. Neck veins were distended and the venous pressure in the antecubital vein was 24 cm. of saline. A portable film of the chest was interpreted as showing an increase in the cardiac shadow. Following intravenous therapy the blood pressure rose to 68/58, but soon dropped to 45/20. After two and a half hours' treatment by fluids and stimulants, her condition became progressively worse and a diagnosis of stab wound of the heart with cardiac tamponade was made.

Under nitrous oxide-oxygen positive pressure anesthesia, the chest was opened through a Spangaro incision. A perforation into the chest cavity was found in the fourth interspace about 3 cm. lateral to the sternum. Through the laceration blood spurted with each expiration. On resection of the third and fourth costal cartilages the blood was found to be coming from both ends of a severed internal mammary artery. There was a hematoma in the adjacent tissue. A large dilated pericardial sac bulged into the operative field. Immediately on opening the pericardium the blood pressure rose from 80/50 to 130/90 and remained stable. No injury to the heart was found. The heart was observed for some minutes before closing. The pericardium was approximated without difficulty, leaving a decompression opening into the left chest cavity at one end of the incision. The patient returned to the ward with a blood pressure of 140/80 and a venous pressure 10 cm. of saline. Recovery was uneventful.

The original interpretation of this case was that external pressure from a hematoma was causing the tamponade. On looking back over the experience and in view of these subsequent cases, I do not believe that hematomatous infiltration was responsible. Certainly no sizable quantity of hematoma was evacuated.

It is probable that alcohol and trauma were the major etiologic factors causing dilatation in Cases I and III. In Case II the dilatation followed anoxemia of the cardiac

muscle resulting from the coronary obstruction. Once complete cardiac failure starts, a vicious circle must take place, since the poorer the circulation the less the oxygen supply, and with the anoxemia the heart muscle loses its tone. The limits of dilatation are reached with the limits of elasticity of the pericardial sac. When the heart can no longer dilate, the flow of blood into the heart chambers ceases; since the muscle fibers are no longer on a tension, they lose their contractility and cardiac activity stops.

Beck and Bright[10] tried to produce rupture of the heart in dogs by sudden compression of the abdomen and hind extremities, thus creating a suddenly increased volume in the vascular bed above the diaphragm. They found that in animals with an intact pericardium the heart dilated to a certain maximum regardless of additional increase in compression. The average increase in the transverse cardiac *x*-ray shadow was 2.3 mm. Another group of experiments was run in which the pericardium had been split from base to apex prior to compression of the abdomen and legs. The average increase in the transverse diameter of the cardiac shadow of this series of animals was 9 mm. In their conclusions and summary, Beck and Bright state, that "the pericardium supports the heart as it dilates in response to this sudden alteration in the circulation, was shown by experiment." Also, "the pericardium may restrict acute dilatation of the heart."

That the pericardium does restrict acute dilatation of the heart, there can be little doubt. But there can be considerable doubt that this restriction on an acutely failing heart is entirely beneficial, since it is probably another cause of an acute cardiac compression syndrome.

## SUMMARY

1. Three explored cases of cardiac compression resulting from acute cardiac dilatation are reported.

2. Acute coronary occlusion from ligation of the left coronary artery, with sequence of events is described.

3. A new point in the pathologic physiology of the heart and pericardium during acute cardiac dilatation is presented.

## REFERENCES

1. Beck, C. S. Two cardiac compression triads. *J. A. M. A.*, 104: 714–718 (March 2) 1938.
2. Elkin, D. C. The diagnosis and treatment of wounds of the heart. *J. A. M. A.*, 111: 1750–1753 (Nov. 5) 1938.
3. Bigger, I. A. The diagnosis of heart wounds. *Southb. M. J.*, 29: 18–23 (Jan.) 1936.
4. Cox, D. M. Wounds of the heart: report of two cases. *Arch. Surg.*, 17: 484–492, 1928.
5. Mayer, J. M. The clinical management of injuries to the heart and pericardium. *Surg., Gynec. & Obst.*, 62: 852–864 (May) 1936.
6. Griswold, R. A. and Drissen, E. M. Wounds of the heart. *Kentucky State M. J.*, 34: 471–474, 1936.
7. Maguire, C. H. Personal communication.
8. Griswold, R. A. Chronic cardiac compression due to constricting pericarditis. *J. A. M. A.*, 106: 1054–1057 (March 28) 1936.
9. Mautz, F. R. Reduction of cardiac irritability by the epicardial and systemic administration of drugs as a protection in cardiac surgery. *J. Thoracic Surg.*, 5: 612–628 (Aug.) 1936.
10. Beck, C. S. and Bright, E. F. Changes in the heart and pericardium brought about by compression of the legs and abdomen. *J. Thoracic Surg.*, 2: 616–627 (Aug.) 1933.

# ABDOMINAL ACTINOMYCOSIS

## RECOVERY FOLLOWING SURGICAL TREATMENT AND USE OF ZINC PEROXIDE AND SULFANILAMIDE

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THE usual methods of treatment of actinomycosis have included the use of potassium iodide, thymol, *x*-ray and copper sulfate, and of surgical drainage. Recently, treatment with sulfanilamide has been recommended.[1] In one case we employed not one agent but a combination of several agents: first, adequate surgical drainage, all the sinus tracts being carefully curetted and exposed widely but never beyond the area of induration; second, "effective" zinc peroxide, as advocated by Meleney for retarding the growth of the potentially anaerobic actinomyces, and, third, sulfanilamide.

### CASE REPORT

A man 45 years of age, a butcher, entered the Evanston Hospital on December 26, 1938, with the complaint of a loss of 45 pounds and soreness in the right side of the abdomen for four months, associated with swelling for two weeks. Two years previously he had been treated for dysentery and one year previously for anal fissure. Four months previously hemorrhoids were given injection treatment. He had enjoyed good health until four months prior to admission to the hospital, when soreness in the right side caused considerable discomfort, especially when he was walking or was lying on that side.

Examination revealed that immediately beneath the skin there was a firm, fixed, irregular nodular mass about the size of a grapefruit. This mass was definitely inflammatory in nature and had caused the overlying skin to become reddened and hot. There were no sinuses in the skin. The mass was dull on percussion, did not pulsate and seemed to be located outside the abdominal cavity. Rectally there was found a moderate amount of induration on the right side. The temperature was 100.4°F., the hemoglobin value 8 Gm. and the red blood cell count 3,600,000. Roentgen study of the gastrointestinal tract disclosed no abnormality.

A preoperative diagnosis of actinomycosis of the abdominal wall, probably originating from the ileocecal region, was made. At operation the diagnosis was confirmed by discovery of ray fungi in the pus on microscopic study. The sinuses were curetted carefully. The cavity was then packed with vaseline gauze, which held several Carrel tubes in place. Through these tubes, solution of zinc peroxide was injected. The dressings were changed daily, and fine-mesh gauze impregnated with zinc oxide ointment was packed into the wound. Through the Carrel tubes zinc peroxide was injected at intervals of two or three hours. On the second day after operation the patient was given four doses each of 20 gr. of sulfanilamide. The treatment was continued for nine days and was stopped because of nausea and vomiting. However, after the patient recovered from this reaction, the drug was again given for a period of four days in the same amounts. Healing of the wound progressed nicely. During the postoperative course pneumaturia developed, and cystoscopic study revealed a 0.6 mm. red edematous area without any demonstrable opening. No evidence of cystitis was found. Culture and smear of the urine showed no evidence of sulfur granules. It was believed that the process was secondary to the actinomycosis. The condition improved without becoming serious.

The abdominal wound was clean and was healing well when the patient was discharged, twenty-seven days after operation. Two months after discharge he was in good health and had returned to work. The wound was practically healed.

We believe that this case illustrates a satisfactory method of treatment of abdominal actinomycosis. The essential factors are: first, the early recognition of the condition and, second, the intensive treatment by means of (a) thorough surgical drainage and curettement; and (b) the use of zinc peroxide dressings; and (c) sulfanilamide.

REFERENCES

1. Walker, O. Sulfanilamide in the treatment of actinomycosis. *Lancet*, 1: 1219, 1938.
2. Miller, E. M., and Fell, E. H. Sulfanilamide therapy in actinomycosis. *J. A. M. A.*, 112: 731, 1939.

# CORRECTION

Dr. R. J. Behan, author of an article entitled "The Treatment of Suppurative Generalized Peritonitis with Alcohol," which appeared in our October issue, wishes to make a few changes in his statements.

On page 97, the last paragraph, first column, should read: "In this period from 1920 to 1931 we had forty-six cases of generalized peritonitis, forty-two resulting from appendicitis and four resulting from other causes in which treatment with alcohol was given." In the same paragraph, second line from the top, right hand column, the per cent of mortality should read 5.79 and not 6.15; and on page 98, third line, right hand column, the death rate should be 5.79 instead of 8.69.

[From Fernelius' Universa Medicina, Geneva, 1679.]

# BOOKSHELF BROWSING

## THE PRACTICE OF OBSTETRICS FIFTY YEARS AGO (1890)

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WHEN The Brooklyn Gynecological Society was formed fifty years ago, there was perhaps some discussion as to whether it should be called gynecologic or obstetric. I do not know. I am certain, however, that Brooklyn gynecologists have always held obstetrics as their major interest. And this is as it should be.

Obstetrics with its roots deep in anatomy and physiology, biology and biochemistry grows at a great pace in our day. There are many living today who have contributed to the development of prenatal care itself, intravenous dextrose therapy, blood transfusion, a more rational approach to the problem of eclampsia, consistent improvement in the technique of cesarean section, accurate pelvic roentgenography and finally our new appreciation of the importance of droplet infection.

Nothing is older than birth. Delivery, whether it was accomplished or managed, has always been a problem; yet no real advance was made from the time of Soranus in the second century until Parè revived podalic version some 1500 years later. There was no textbook in any language for 1400 years. In the sixteenth century the remarkable folklore of the past was copied and printed to serve as a textbook for all the physicians in Europe for another 200 years.

We have no lack of textbooks, but even in 1890 they were few. The English textbook of Playfair and "The Science and Art of Midwifery" by William Thompson Lusk, Professor of Obstetrics and the Diseases of Women and Children in the Bellevue Hospital Medical College were easily the most popular. Lusk's text was used in the Long Island College Hospital, the University of the City of New York, the College of Physicians and Surgeons and the New York Polyclinic as well as in the author's own school. In the edition of 1890, dedicated to Fordyce Barker "In grateful acknowledgment of his generosity toward the younger members of his profession" we should be able to discover the obstetrical teaching of that day.

In the preface we read, "It is with special gratification that I take this opportunity to draw attention to the triumph of the modern doctrines concerning the nature of puerperal fever, and to the wonderful results that have proceeded from the application of Mr. Lister's teachings to the hygiene of lying-in hospitals. The latter have ceased to be pest-houses, and it is now a question whether forlorn women confined in properly conducted maternities, are not actually safer than the fortunate classes in their own homes."

His discussion of the hygiene of pregnancy is good, yet his admonition to wear flannel drawers would hardly meet with feminine approval today. "Since the sobering effect of experience has invariably served to dispel illusive hopes . . . it is usually therefore the part of prudence to do nothing for morning sickness or even continuous nausea, . . . to let the wife go home on a visit to her mother furnishes a serviceable hint in the way of practice." For continued vomiting, he advised that the cervix, if eroded, should be brushed with 10 per cent nitrate of silver, or the faradaic current or an ether spray applied to the pit of the stomach, or an icebag to the cervical vertebrae. W. Gill Wylie, he states, has found that most cases of nausea were cured by opening the cervix with a dilator of his own invention, dusting the cervix with iodoform and applying a glycerin tampon. Dilatation was effected "without much risk" up to the internal os; even when the cervix was so patulous that dilatation seemed useless, stretching of tight bands found well up in the cervix brought prompt relief. Rarely were more than two treatments necessary.

In labor the physician was advised to repeat his vaginal examination at hourly intervals during the first stage and frequently in the second. If rupture of the perineum was felt to be imminent, "mock modesty should be discarded, and the parts imperiled should be unhesitatingly exposed to view . . . the physician is justified in making lateral incisions through the vulva . . . to this operation the term episiotomy is applied, yet it should never be performed so long as hope exists of otherwise preserving the perineum. It is essentially the operation of young practitioners, the occasions for its employment diminishing in frequency with increasing experience." The operation was lateral, done with a bistoury so as to divide the vagina and not the skin. The abdominal binder, he thought, should not be discarded as some had advised, yet to apply it "with address is not an indifferent accomplishment."

Lusk knew only ether and chloroform and advised caution in their use, for "the practice admits neither of ignorance or carelessness. There is no doubt that the vast majority of medical men refrain from the use of anesthetics in ordinary labor, either from vain apprehensions, or because some incident in their practice has led them to suspect that, in spite of statistics, they are not devoid of objectionable or dangerous properties."

"The diagnosis of multiple pregnancy," he writes, "is rarely to be made out with absolute certainty." If delivery of the second child were delayed more than an hour, the membranes should be ruptured, but "In case of a premature child delivered with its own placenta, cases of continued development in utero of the remaining child would point to the policy of abstention."

Breast feeding was advised. "If it is impossible to procure the services of a wet nurse, or if the aversion of the parents to wet nurses as a class proves unconquerable, artificial alimentation must be tried. It is unquestionable that many babies thrive fairly when brought up on the bottle."

In the treatment of hydatidiform mole, after its expulsion or manual removal, the uterus was irrigated or swabbed with perchloride of iron; the Thomas' dull-wire curette was used for persistent hemorrhage. "Its prognosis is not extremely unfavorable in the majority of cases," he writes, and "determined chiefly by the violence and frequency of the attending hemorrhages." In the treatment of abortion, repeated solid and effective packs and manual removal were in general use, with sponge tents, when necessary, for dilatation of the cervix. Lusk liked to use a tolerably firm curette where "owing to inflammatory conditions, I have hesitated to make the circuit of the uterine cavity with my finger."

In 1890, the diagnosis of extrauterine gestation before rupture was rarely made; death often occurred without the slightest suspicion as to the nature of the case. We read: "The indication for treatment in the early months is plainly the adoption of

measures to destroy the life of the fetus, and thus by arresting the growth of the ovum, avert the danger of rupture and hemorrhage. . . . Spontaneous recovery commonly follows the death of the embryo." Puncture of the sac through vagina or rectum, or injection of morphin or atropin into it were in common use. T. Gaillard Thomas had opened the sac with the thermocautery and the patient narrowly escaped with her life. Lusk thought that daily use of the faradaic current, with one pole in the rectum or vagina and the other over Poupart's ligament was safe and efficient. This treatment was popularized by Thomas who saw most of the cases in New York in consultation. He was aware of Lawson Tait's success with laparotomy, but apparently had no experience with it. At operation it was advised, when the peritoneum was reached, that a small nick be made first and a small pipette introduced for verification of the diagnosis. He writes, "Strange to say, but two operators in these days of abdominal surgery have had the hardihood to carry these (instructions) into execution. The reasons for this backwardness are probably to be found in the uncertainties of the diagnosis, the risk of finding the sac hopelessly matted to the adjacent viscera, the dislike of operating upon a dying woman, and the fact that a considerable number of spontaneous recoveries occur." Playfair thought it "A bold and heroic procedure, not hopeless, since the alternative is death."

Because of its hazards, cesarean section was "chiefly justifiable in cases in which craniotomy and the delivery of the child through the natural passages involve the life of the mother in still greater peril. It is indicated, therefore, in extreme pelvic contraction, in the case of solid tumors which encroach upon the pelvic space and in advanced carcinomatous degeneration of the cervix."

The operating room should be thoroughly aired and disinfected by burning sulfur and "a convenient operating table can be readily improvised by covering an ironing table with blankets or an old-fashioned comforter." The intestines were outlined by percussion and pushed to one side out of harm's way. An assistant, whose principal function was to guard against protrusion of the intestines, steadied the uterus in the midline as the incision was made; when the uterus was exposed, he redoubled his vigilance, compressing the uterus firmly against the incision, dextrously hooking his index fingers into the angle of the incision and lifting the uterus into close contact with the abdominal wall to prevent escape of amniotic fluid into the peritoneal cavity. The incision was then enlarged with a bistoury and "the membranes ruptured through the wound, rather than through the vagina; it is more quickly effected, and time at this stage is precious . . . even where, at the conclusion of the operation, the uterus is well retracted and the hemorrhage arrested, it is well to still use the sutures as a prophylactic against secondary hemorrhage." Some operators were content with friction and the use of ice, but Lusk used carbolized silk sutures since catgut had been found impracticable; the knots loosened with postpartum contractions of the uterus.

"It is obvious," says Lusk, "that the classical cesarean section, even with the addition of the suture and the employment of strict antisepsis, is still far from answering the requirements of a conservative procedure," since separation of the uterine wound occurred in spite of the suture. He knew that Kehrer had, in 1882, opened the uterus through a transverse incision in the lower segment, separating the peritoneum so as to cover his muscle sutures with it. The cesarean operation in this country was formidable with about 60 per cent mortality. The results of laparo- or gastro-elytrotomy, the extraperitoneal operation devised by Thomas, and successfully performed by Skene and Jewett in 1885 were such "as to inspire a confidence in its feasibility" at least equal to that employed by the rival procedure of Porro.

Lusk's attitude toward induction of labor when eclampsia threatened was no different

from ours, yet he said that the weight of authority was for procrastination, "the interruption of pregnancy being regarded an extreme measure, justifiable only in cases of utmost peril." He advised phelbotomy of eight to sixteen ounces as the first step in the treatment of convulsions.

Most authorities advised expectancy in the treatment of the hemorrhages late in pregnancy, except when the loss of blood was alarming, but Lusk taught that there was no safety as long as pregnancy continued and advised Braxton-Hicks version or a bag.

"The treatment of postpartum hemorrhage," he said, "is one of the most satisfactory departments of obstetrical practice." Transfusion is theoretically rational.

"In practice, however, the difficulties of the technique, the hesitation of the bystanders to furnish the required blood, combined with the somewhat unsatisfactory results of transfusion are all obstacles to its employment. For a long time I carried with me Aveling's very simple and ingenious transfusion apparatus. In the few cases where I had intended to use it, however, the tubing was never in perfect order. Professor Gaillard Thomas recommends the substitution of the intravenous injection of milk for that of blood" as a perfectly feasible, safe and legitimate procedure. "None but milk removed from a healthy cow within a few minutes of the injection should be employed. Decomposed milk is poisonous and should no more be used than decomposed blood. The intravenous injection of milk is infinitely easier than the transfusion of blood . . . and like that of blood is commonly followed by a chill, and rapid and marked rise of temperature; then all subsides, and great improvement shows itself in the patient's condition. Not more than eight ounces of milk should be injected at one operation." Playfair advised the use of defibrinated blood, but said that there were no encouraging statistics and that transfusion should be done only when life was on the verge of extinction.

It is good for us to review the errors of the past, not for the cultural value alone, but to remind us that we, too, may be just as far from the truth. I venture to say that the gap between our knowledge and the future is far greater than that which separates us from all the past.

SPECIAL MONOGRAPH

# Diagnosis and Treatment of Various Types of Diaphragmatic Hernia

BY
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AMERICAN JOURNAL OF SURGERY, Inc.
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# DIAGNOSIS AND TREATMENT OF VARIOUS TYPES OF DIAPHRAGMATIC HERNIA*

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THE most common lesions of the diaphragm which require surgical treatment are abnormal openings through its structure which permit herniation of the abdominal viscera into the thoracic cavity. These abnormal openings may be attributable to congenital structural deficiencies of the diaphragm and occasionally of the esophagus, or they may be caused by traumatic rupture or inflammatory necrosis of the normal diaphragm.

The subject of the diagnosis and treatment of diaphragmatic hernia has received more consideration in recent years because its more frequent recognition has changed its status from that of a rare condition to one that is not infrequently encountered. However, I believe that this subject deserves more general interest and consideration than it is accorded at present, since in many instances the diagnosis is not made until complications have occurred. This subject is of interest to the clinician because the diagnosis is of first importance, the symptoms are often complex and the condition frequently must be considered in the differential diagnosis of diseases of the upper part of the abdomen and lower part of the thorax. It is of interest to the roentgenologist because the roentgenologic recognition of diaphragmatic hernia is often the only means by which a definite diagnosis can be established clinically. The treatment is of primary concern to the surgeon because operative replacement of the herniated viscera, and repair of the abnormal opening in the diaphragm are the only treatments that promise complete relief of symptoms to the patient.

## EMBRYOLOGIC ASPECTS

The formation of the diaphragm from embryonic structures is a highly complex process, because its muscular elements are derived from several sources.

The anterior, lateral and central parts, which comprise the greater portion of the diaphragm in the adult person, are formed from the transverse septum and fused ventral mesentery. The

* From the Division of Surgery, The Mayo Clinic.

remaining posterolateral portion is formed by the fusion of the dorsal mesentery and the mesoderm derived from the receding wolffian body and the pleuroperitoneal membrane derived from the pulmonary ridge. It is difficult to determine the exact amount of the muscle tissue that is derived from each of these structures, since there is probably considerable variation, but it is probable that the dorsal mesentery forms the posterior and central portions containing the esophageal opening. The mesodermic cells from the receding Wolffian body form the right and left crura. The pleuroperitoneal membrane grows ventrally and closes the remaining opening (hiatus pleuroperitonealis) between the peritoneal celom and the pleural celom, by fusing with the transverse septum, and forms the lateral portion of the diaphragm. Failure of fusion or proper deposition of the mesoderm at any one of these adjacent points of union may result in congenital continuity of the pleural and peritoneal cavities or a congenitally weak portion in the diaphragm at any of these points. Consequently, from an embryologic standpoint, weak portions might be expected to appear at the point of fusion of these different structures. These portions are situated dorsolaterally at the fissura pleuroperitonealis (foramen of Bochdalek); also through the outer crus and through the esophageal opening. Hernia through the dome is common but cannot be explained on the foregoing basis, because it is not a fusion region. Such a hernia may be the result of excessive degeneration of the muscle in the formation of the central tendon or of some pathologic condition. Unilateral absence of the diaphragm probably is the result of the failure of development of the pleuroperitoneal membrane, which usually is found as a narrow ridge of tissue along the posterior wall of the thorax.

*Congenital or Embryologic Aspects of Nontraumatic Hernias and Diaphragmatic Defects.* Diaphragmatic congenital defects and nontraumatic hernias are variously reported as being from ten to eighteen times as frequent on the left side. The cause undoubtedly is to be found in the embryonic formation of the diaphragm. This point is a subject of controversy among embryologists. I have attempted to show the different stages in the formation of the diaphragm in embryos of various sizes; but in order to arrive at any satisfactory conclusions as to why hernia is more likely to occur in these regions, one must not only follow the concomitant changes that are occurring in the developing embryo, but must also consider the

physiologic manifestations which are present in the constantly changing organism at the same time.

The septum transversum, from which the anterolateral portion of the diaphragm of the adult person is formed, arises from the mesoderm in the upper part of the cervical region. Its descent is gradual in the early embryonic weeks, and at the 5 to 6 mm. stage it is opposite the fourth and fifth cervical segments, where it receives its innervation from the branches of the third, fourth and fifth cervical nerve roots to these segments (the phrenic nerve). At approximately the same period as that in which the septum transversum appears (2 to 3 mm.), the tracheal bud appears from the ventral surface of the esophagus, and the liver buds have grown vertically into the under surface of this descending septum. The anlage of the stomach also has appeared as a definite thickening of the foregut opposite the fourth cervical segment.

Three important developmental processes occur in embryos of the 5 mm. stage. The pulmonary ridge appears as a definite shelf of mesodermal tissue which forms the pleuropericardial and pleuroperitoneal membranes. The former closes off the pericardial cavity from the pleural cavity at about the 10 to 11 mm. stage, and does not enter into the formation of the diaphragm. The latter ultimately will help to fill in the posterolateral portion of the diaphragm. At this stage (5 mm.) the pleuroperitoneal membrane is only a narrow shelf of mesodermal tissue that projects into the celom, marking the caudal dividing line of the pleural and peritoneal cavities, which are at this stage in free communication with each other.

The second noteworthy change is the unsymmetric development of the right and left pulmonary buds, which are at a slightly lower level than the septum transversum. The right bud grows caudally and posteriorly and an infracardiac bronchus soon develops. The left bud grows almost transversely to the vertical plane and, because of the presence of the left enlargement of the heart, an infracardiac bronchus does not develop. This process places the left lung at a somewhat higher level than the right lung.

The third change occurring at this period is the increased activity in the development of the midgut, which previous to this period has been practically a straight tube. It begins to elongate, with an angulation toward the umbilicus from a cephalic and caudal limb of this portion of the intestinal tract.

From the foregoing it is seen that at this early period of the embryo, the cavity of the body is beginning to divide into two main cavities, the peritoneal cavity and the pleural cavity, by an incomplete shelf of fetal muscular tissue derived from several sources. Ventrolaterally, this shelf is called the "transverse septum"; dorsally, the less well developed mesodermal shelf is called the "pleuroperitoneal membrane." The intestinal tract with its dorsal mesentery and the Wolffian body extends into the celom in a dorsomesial position. At this stage, there is free communication between the two cavities; this opening may be termed the "pleuroperitoneal hiatus."

In the 7 mm. embryo, the transverse septum with the pleuroperitoneal membrane begins to migrate caudally, rapidly passing the pulmonary buds and the anlage of the stomach, placing the greater portion of the lung in the pleural cavity and the anlage of the stomach partially above the diaphragm.

In the 9.4 mm. embryo, the liver has become firmly anchored to the right dorsal wall of the body of the plica vena cava. The midgut begins to grow more rapidly and the yolk sac disappears. This action frees the intestine from the umbilicus, and rotation of this segment of bowel begins.

In the 11 mm. embryo, the stomach, which has been practically stationary, now follows rapidly behind the descending septum transversum, and in the 17 mm. embryo it has virtually reached its permanent position. This descent is made possible by the sudden elongation of the esophagus. During this descent the right and left dorsopleural recesses are converted into bursae which surround the cardia. The left bursa usually disappears, and the right bursa, when well developed, is known as the "infracardiac bursa" and bears a definite relationship to esophageal hernia. At about this period (11 mm.) a physiologic alteration in the abdominal region takes place. The celom has not increased in size enough to accommodate the rapid changes that are taking place within its walls and which are augmented by the descent of the septum. As Mall stated, "Since the liver grows downward and crowds upon the rapidly elongating intestine, the intestine must escape it if it has a chance and the coelomic space within the umbilical cord naturally receives it." This phenomenon, known as "physiologic umbilical herniation," provides a reservoir for the abdominal contents until the peritoneal cavity has grown sufficiently to store its own contents. The herniated

intestine is restored to the abdominal cavity by the growth of the abdominal wall past the fixed mesentery of the intestines. This physiologic herniation starts in the 11 mm. embryo, is well developed in the 22 mm. embryo and starts on its way toward voluntary reduction in about the 35 mm. embryo.

During this period of greater activity in the abdominal celom, the diaphragm is incompletely formed, and the hiatus pleuroperitonealis is patent. The closure takes place on the right side first in about the 17 or 18 mm. embryo and on the left side in about the 19 or 20 mm. embryo. The left side of the liver has largely disappeared, and the whole organ has rotated to the right and fused with the right wall of the body, protecting to a great extent the right half of the diaphragm and probably aiding its most rapid completion. The stomach has reached its permanent site below the diaphragm before the left half of the diaphragm is closed. Therefore, the hiatus pleuroperitonealis patent on the left side is in direct contact with the rapidly forming and constantly changing hollow viscera during this period of physiologic umbilical herniation.

It would seem that this time of the incomplete closure of the diaphragm in relationship to multiple rapid changes which are taking place in the embryo is of great importance in the preponderance of left-sided congenital defects. To recapitulate, these changes consist of: (1) the occurrence at this time of the normal physiologic umbilical herniation; (2) the patency of the hiatuses connecting the peritoneal and pleural cavities; (3) the sudden descent of the diaphragm, which is considerably in advance of the descent of the stomach; (4) the shift of the liver to the right because of anchorage and the degeneration of the left lobe from vascular change and pressure; (5) the presence of hollow mobile viscera on the left side; (6) the elevated position and smaller size of the left lung; (7) the rotation of the stomach to the left, and (8) the presence of bursae at the esophageal opening.

On the basis of the foregoing material it is seen that embryonic herniation would occur most commonly in the 11 to 20 mm. embryo. Congenital herniation of somewhat later formation can be explained on many of the same grounds. It is the result of failure of parts of the diaphragm to mature, or of excessive degeneration of muscular elements in the formation of the central tendon.

Inasmuch as the stomach descends behind the septum transversum, if there is a delay in descent of the stomach, the lumbar

portion of the diaphragm will be imperfectly developed, and the esophageal hiatus will be formed around the cardiac end of the stomach instead of around the esophagus. This will result in an abnormally large hiatus with deficiency both in the muscle ring and in the attachments of the diaphragmatico-esophageal membrane. The degree of the deficiency depends on the amount of gastric anlage in the thorax at the time of muscularization of the lumbar portion of the diaphragm. In rare instances the stomach may remain in the elevated position as a result of a congenitally short esophagus and a partial thoracic stomach will result. In most instances the esophagus continues to elongate normally, thus placing the stomach below the diaphragm. This abnormal position of the stomach will cause an abnormal enlargement of the esophageal hiatus to be produced, with a wide space between the margin of the muscle and the wall of the esophagus. It will also result in imperfect fixation of the elastic membrane to the esophagus and stomach and an abnormally large peritoneal fold extending well down on the cardia of the stomach. This abnormal relationship will permit much more flexibility of the esophagus in the enlarged hiatus than is normal. The more defective the formation of the hiatus, the more likely is herniation of the stomach to occur through it in later life. The true congenital hernia present at birth may be explained in the same way, but it is probable that persistence of the dorsopleural recess may be a factor in the origin of congenital hernias.

## ANATOMIC ASPECTS

The diaphragm is a single, independent, dome-shaped muscle arising from the circumference of the lower part of the thorax and which, when it is normally formed, completely separates the abdominal and thoracic cavities. (Fig. 1.) The muscular structure of the diaphragm in the adult person is divided into three portions according to the origins of these portions: the sternal, costal and lumbar portions. The sternal is the weakest of these portions and the lumbar is the strongest. All three portions are inserted into the margin of a central tendon. The sternal portion consists of a few slender fasciculi arising from the posterior surface of the xyphoid cartilage. There are muscular deficiencies on each side of the cartilage filled with areolar tissue and covered with pleura and peritoneum through which the superior deep epigastric vessels pass. These deficiencies are called the foramina of Morgagni or Larrey's spaces.

The costal portion forms the main part of the dome of the diaphragm and arises by broad bands of muscle from the lower six costal cartilages and from the eleventh and twelfth ribs, interdigitating

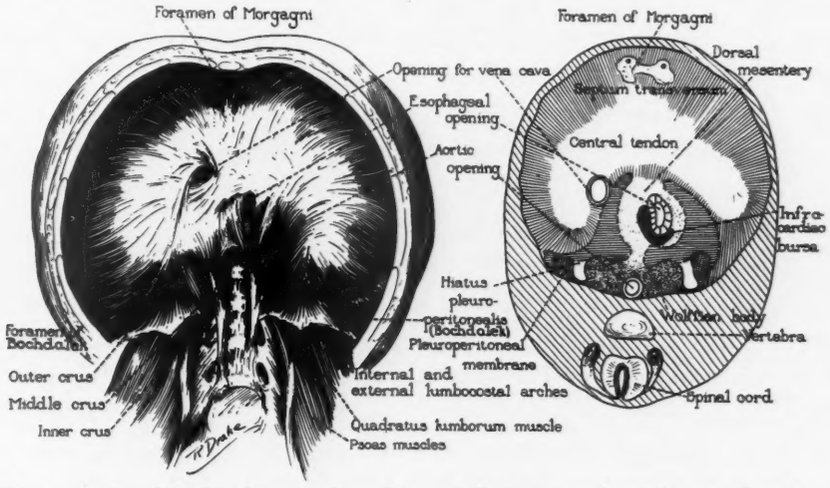


FIG. 1. Left, adult diaphragm viewed from below; normal openings and potential sites of herniation. Right, semidiagrammatic reconstruction of embryonic (17 mm.) diaphragm showing the embryonic structures concerned in the formation of the adult diaphragm and the sites for possible congenital deficiencies. This diaphragm shows the hiatus pleuroperitonealis patent on both sides at this stage. The space enclosed by the dotted lines indicates the extent of this opening at the 11 mm. stage. The infracardiac bursa, the potential site of herniation around the esophagus, is also shown. (*Arch. Surg.*, 16: 386, 1928.)

with the transversalis muscles. This portion covers the greatest area. There often are areas devoid of muscular tissue situated between the individual bands that arise from the wall of the thorax.

The lumbar portion arises from the bodies of the lumbar vetebrae and consists of three crura on each side: the inner, middle and outer crura. The inner crus is much the strongest of the group, and arises from the anterior surface of the third and fourth lumbar vertebrae. The greater portion of the inner crus is muscular, but the inner margins soon become tendinous and unite at the level of the twelfth thoracic vertebra. This arch, with the bodies of the twelfth thoracic and first lumbar vertebrae, forms a canal for the passage of the aorta and thoracic duct. This is called the aortic opening; it is in reality not an opening through the diaphragm, but is situated entirely behind the diaphragm. The esophageal opening is anterior to, and a little to the left of, the aortic opening. It is entirely surrounded by muscular tissue and is formed by the decussation of the

crura on a level with the tenth thoracic vertebra. It transmits the esophagus and its vessels and the two vagi nerves. The middle crus arises from the lateral surfaces of the body of the second lumbar vertebra; it is separated posteriorly from the inner crus by narrow slits which transmit the greater and lesser splanchnic nerves of each side, and the left crus transmits the vena azygos minor. The vena azygos major transverses either the aortic opening or the right crus. The outer crus arises from the internal and external lumbocostal arches (arches of Haller) which arch over the psoas and quadratus lumborum muscles posteriorly.

The lateral lumbar portion of the diaphragm arises chiefly from the inner lumbocostal arch, with only a few fibers arising from the external lumbocostal arch. There is usually a triangular-shaped space of muscular deficiency between the outer crus of the lumbar portion of the diaphragm, the last muscular part of the costal portion of the diaphragm, and the twelfth rib. The apex of the triangle is curved upward and forward toward the tendinous portion of the diaphragm. The base is turned downward and somewhat backward and partly rests on the twelfth rib, but sometimes it extends in front of the body of the psoas muscle. The borders of this hiatus are muscular and are more or less of sphincter-like construction. The space is closed by a membrane which consists of two sheaths; the upper sheath comes from the pleura, and the lower sheath is thin and is a continuation of the iliac fascia. This space was first described by Bochdalek, in 1848, and has been named the "foramen of Bochdalek."

The central tendon of the diaphragm is reniform, with the central portion slightly curved and extending into the dome, of which the greater portion is on the right side. At the base of this right portion and entirely within the tendon is a large quadrilateral foramen which gives passage to the inferior vena cava. This opening is on a level with the disk between the eighth and ninth thoracic vertebrae.

The diaphragm receives its motor innervation from the phrenic nerve, which originates from the fourth and sometimes through branches from the third and fifth cervical nerves. This cervical origin of the innervation of the diaphragm is due to the embryonic derivation of the diaphragm from cervical myotomes which are innervated by the third, fourth and fifth cervical nerves. These nerves originally may have contained both motor and sensory fibers but most anatomists agree that the phrenic is the chief, if not the only, motor nerve to the diaphragm. The presence of some afferent

sensory nerve fibers in the phrenic nerve will explain the presence of referred pain in the neck which is often associated with disorders of the diaphragm. Some anatomists are of the opinion that the

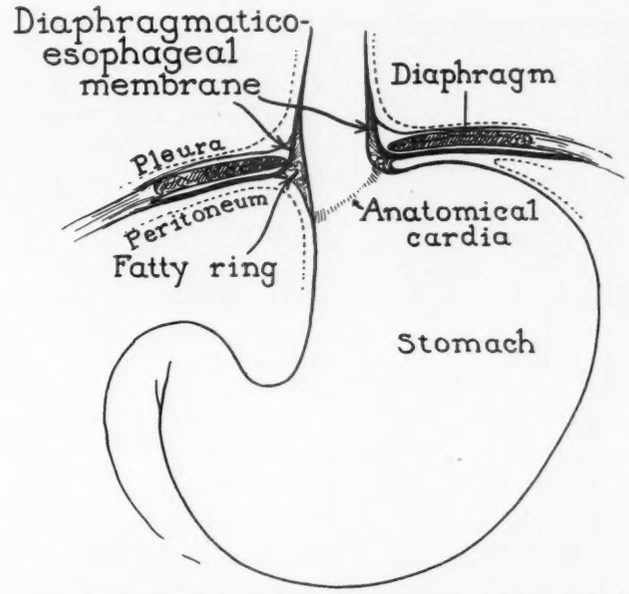


FIG. 2. Normal anatomic relationship between lower end of esophagus, cardiac end of stomach and esophageal hiatus, showing the relationship of the diaphragmatico-esophageal membrane to these structures. (*J. Thoracic Surg.*, 8: 127, 1938.)

peripheral part of the diaphragm receives some motor innervation from the lower intercostal nerves.

The circular muscle bundles of the esophageal hiatus are innervated chiefly by the phrenic nerve of the same side, regardless of whether the muscles originate from the crus of the same side or from that of the opposite side. There also may be some cross innervation from the phrenic nerve on the opposite side.

In considering the normal relationship between the esophageal hiatus and the lower end of the esophagus and cardia, I wish to mention again the fact that the esophageal hiatus is normally situated to the left of the medial line, at the level of the upper border of the tenth thoracic vertebra, in the dorsal part of the dome of the diaphragm.

The muscular elements that form the muscular ring of the hiatus usually originate from the median portion of the right crus, but the hiatus may be formed by the crossing of both right and left crura

after they have united in front of the aorta to form the aortic orifice. This ring of muscle of the hiatus loosely approximates the lower end of the esophagus but is not directly attached to it. Fixa-

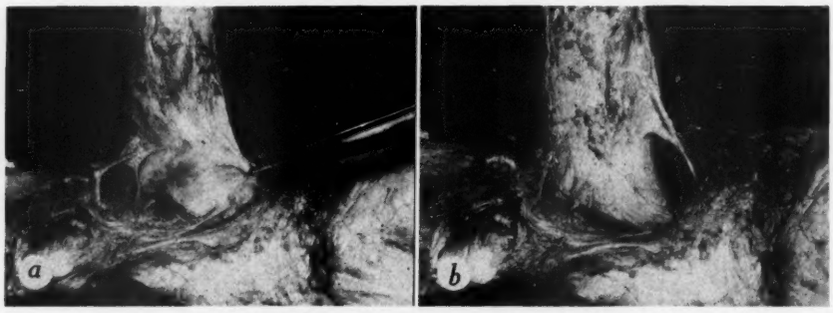


FIG. 3. *a*, dissection of normal attachment of the diaphragmatico-esophageal membrane (held in the clamp). Fascia covering space to right is a persistent infracardiac bursa. *b*, same as *a*, after removal of peritoneum and anterior attachments of diaphragmatico-esophageal ligament showing posterior attachment of this ligament extending beneath and not attached to the circular muscle of the hiatus.

tion of the diaphragm to the lower end of the esophagus and to the cardia of the stomach is obtained by the diaphragmatico-esophageal membrane. (Fig. 2.) This membrane consists of elastic and fibrous tissue originating in the fascial coverings of the diaphragm; mostly those of the abdominal side but partly those of the thoracic side of the diaphragm. (Figs. 3 and 4.) This elastic and fibrous membrane has its origin lateral to the muscular ring of the hiatus over which it passes, with slight attachments, to fill the space between the inner margin of the muscle and the esophagus; it is inserted into the lower 2 or 3 cm. of the esophagus and into the upper 1.5 to 2 cm. of the stomach, in a fanlike manner. This elastic and fibrous membrane constitutes a flexible connection between the diaphragm, esophagus, and cardia, and permits of a certain amount of mobility during the act of swallowing and during respiratory movements. (Fig. 5.) Its greatest strength is in its esophageal attachment, where it acts as an antagonist to the longitudinal muscle fibers of the esophagus, preventing these muscles from pulling the cardia of the stomach through the hiatus. The lower part of the esophagus passes obliquely through the diaphragm and extends below the hiatus for a varying distance of from 0.5 to 1.5 cm., where it joins the stomach at an angle. This junction is the true cardia and normally is always situated below the diaphragm.

The normal peritoneal covering and attachments of the esophagus and cardia of the stomach, around the esophageal hiatus, are of great importance. The abdominal portion of the esophagus is loosely

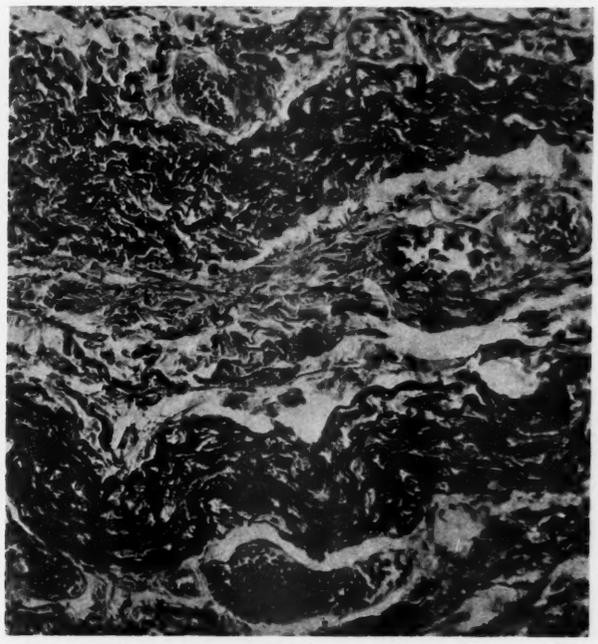

FIG. 4. Photomicrograph of normal diaphragmatico-esophageal ligament removed from previous specimen (Fig. 3*b*), especially stained to show elastic tissue (constituting approximately 50 per cent of the ligament) in regular curved bands between bands of connective tissue.

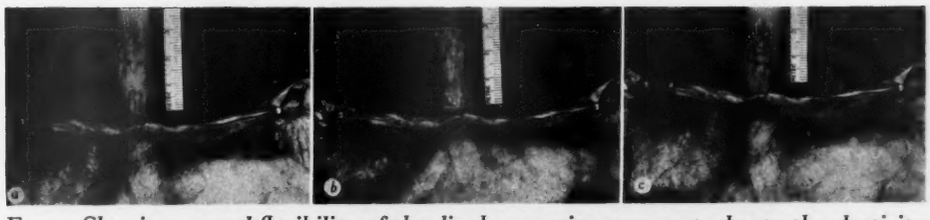

FIG. 5. Showing normal flexibility of the diaphragmatic movements due to the elasticity and flexibility of the attachments of the diaphragmatico-esophageal membrane to the esophagus and stomach: *a*, diaphragm in midposition; *b*, diaphragm in extreme descent *c*, marked elevation of diaphragm.

covered with peritoneum on its anterior and left aspects. (Fig. 6.) The uppermost part of the stomach is loosely covered, for about 2 cm. anteriorly and 3 cm. posteriorly, where the peritoneum fuses with the serous covering of the gastric wall. The union of these two folds of peritoneum forms the gastrophrenic ligament which fixes

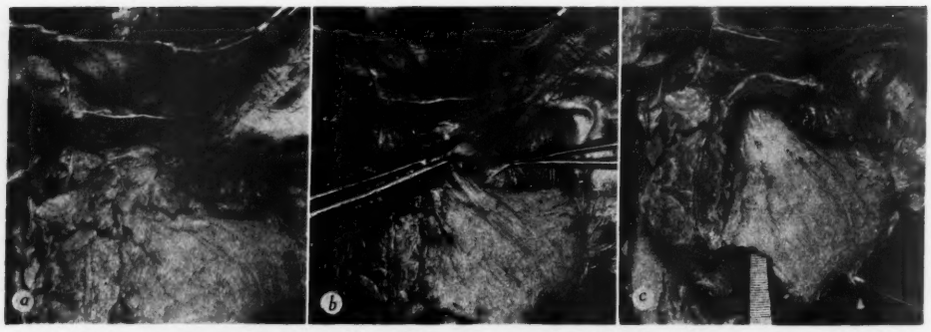


FIG. 6. Showing normal relationship of the peritoneal and subperitoneal fatty pad to the lower part of the esophagus and to the cardia of the stomach: *a*, shows the normal redundancy of the peritoneum; *b*, shows the diaphragmaticogastric attachments of the peritoneum to the cardia and fundus of the stomach; *c*, after removal of the gastric attachment of the peritoneum to the stomach showing 2 to 3 cm. of the esophagus which is normally below the diaphragm.

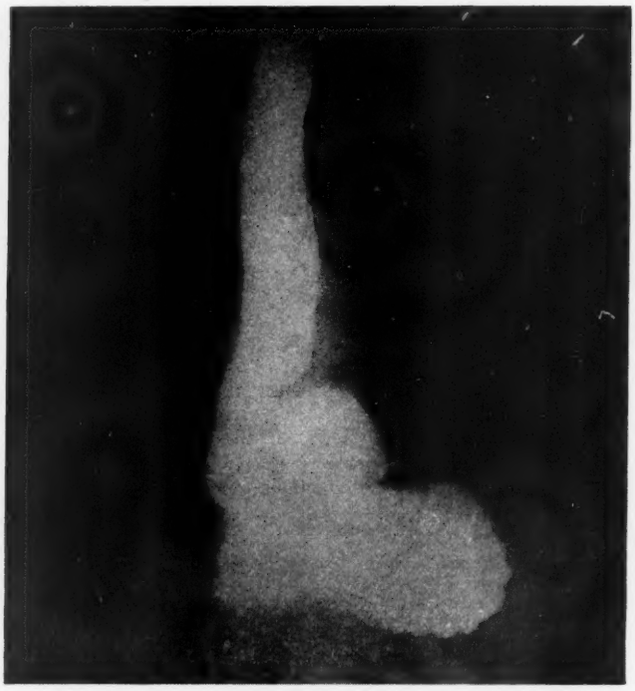

FIG. 7. Roentgenogram on admission of patient showing esophageal hiatus diaphragmatic hernia with herniation of approximately a fourth of the stomach above the diaphragm. Patient aged 65 years; progressive symptoms of four years' duration.

the cardiac end of the stomach to the diaphragm and continues to the left to form the phrenicocolic ligament. Beneath this loose peritoneal covering there is a triangular pad of fat and connective

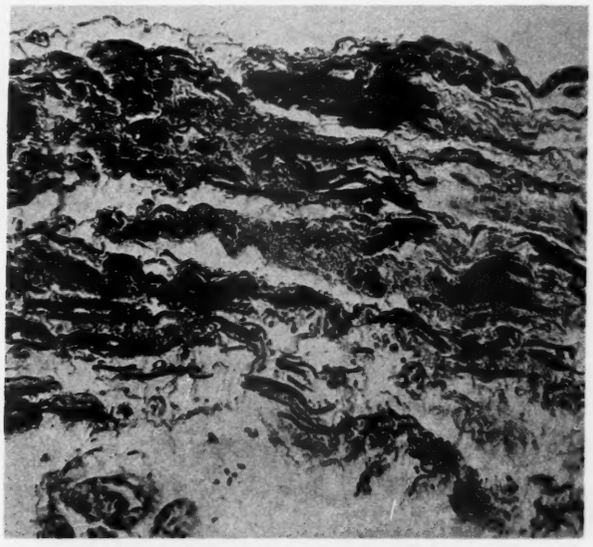

FIG. 8. Same patient as in Figure 7. Photomicrograph of a portion of the diaphragmatico-esophageal membrane which was removed at the time of the operative repair of the hernia, especially stained to show elastic tissue. Atrophy of the elastic tissue bands is evident. (Compare with normal shown in Figure 4.)

tissue and the attachments of the diaphragmatico-esophageal membrane. This loose peritoneal attachment around the esophageal hiatus permits flexible movement of the esophagus without pulling on the peritoneum.

*Anatomic Aspects of Hiatus Hernia.* At this point, a digression may be made to consider the anatomic factors involved in hiatus hernias. The diaphragmatico-esophageal membrane and the loose attachment of the peritoneum at the cardia have considerable bearing on the occurrence of hiatus hernias. It is probable that the hiatus hernias of elderly people, which are accompanied by insufficiency of the hiatus, are attributable to atrophy of this protective elastic membrane, particularly in cases in which there is an abnormally large hiatus. (Figs. 7 and 8.) The elastic membrane has been sufficient during the prime of life to protect the opening but, as the atrophy associated with advancing years develops, the stomach is permitted to protrude through the large hiatus to a position above

the diaphragm. It is probable, in such cases, that this herniation would have occurred earlier in life had abnormal stress been applied, such as trauma, respiratory difficulty or marked increase in intra-

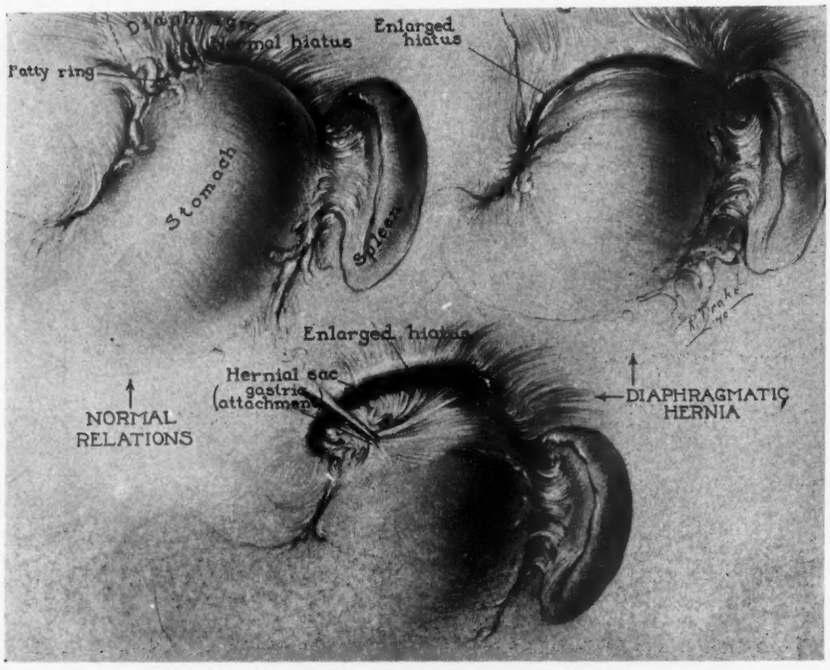


FIG. 9. The anatomic relationships at the cardia of a normal stomach compared to those associated with esophageal hiatus diaphragmatic hernia. These relationships in the latter are shown with the herniated portion of the stomach in the hernial sac and after it has been withdrawn.

abdominal pressure. The elastic and fibrous membrane forms one of the coverings of the hernial sac in the smaller types of hernia. In large hernias this membrane seems to be pushed forward and there is no barrier to the peritoneal sac which enters the posterior portion of the mediastinum. In these cases the entire stomach often herniates into the thorax.

The loose attachment of the peritoneum around the hiatus may be a factor in the formation of hiatus hernias, since it permits the beginning of the herniation of the cardiac end of the stomach and formation of the hernial sac.

In all true hernias this loose peritoneal fold fuses with the serosa farther down on the cardia of the stomach than normal. (Fig. 9.) The peritoneum does not extend around the greater curvature of the stomach to the posterior wall to form the gastrophrenic ligament, but fuses with the serosa of the anterior wall, so that there is no

gastrophrenic ligament and only a very short phrenicolienal ligament. This places the spleen close to the left margin of the hiatus, thus permitting the upper part of the cardiac end of the stomach to be abnormally free posteriorly, beneath the enlarged esophageal hiatus.

The consistency with which the surgeon finds abnormal attachments of the peritoneal fold and of the gastrophrenic ligament to the cardiac end of the stomach, and abnormal enlargement of the muscular ring of the esophageal hiatus, suggests that this particular type of hernia results from a delay in descent of the esophagus as related to the formation of the lumbar portion of the diaphragm.

## CLASSIFICATION OF DIAPHRAGMATIC HERNIA

The term, "diaphragmatic hernia," is commonly used to designate any condition in which there is protrusion of abdominal contents into the thoracic cavity through an abnormal opening in the diaphragm. All true hernias have a sac as one of their component parts, so that many conditions, commonly included under this term but which do not have hernial sacs, would more properly be termed "eviscerations" or "false hernias." The presence or absence of a hernial sac cannot be determined by clinical examination. It can be found only at operation.

There are numerous classifications of diaphragmatic hernia which are based on the embryologic and etiologic aspects, pathologic anatomy, the site of the opening in the diaphragm, the presence or absence of a sac, the content of the hernia and other factors. It is difficult or impossible to make most of these classifications clinically; accordingly, many of them are of little practical value. Diaphragmatic hernias are therefore usually classified in three main groups: (1) congenital, (2) acquired and (3) traumatic.

From a clinical and surgical standpoint, the history of a preceding injury is helpful in establishing the diagnosis and in determining the type, urgency and prognosis of the operative treatment. Because of the practical clinical and surgical significance of trauma as an etiologic factor, I have suggested that diaphragmatic hernia be classified into two main groups: nontraumatic and traumatic. I have subdivided these two groups according to the various types.

*Nontraumatic Hernia.* A nontraumatic diaphragmatic hernia may be congenital or acquired. If it is congenital, the hernia is

attributable to embryologic deficiency and usually does not have a hernial sac. The most common sites of a congenital hernia, in the probable order of frequency of occurrence, are: (1) through the hiatus pleuroperitonealis (foramen of Bochdalek), (2) through the esophageal hiatus, (3) through an anterior substernal opening (foramen of Morgagni) (Larrey's spaces), and (4) through the gap left by partial absence of the diaphragm, a gap which is usually situated in the posterior portion of the muscle.

If the hernia is acquired after birth, the sites of occurrence are: (1) through the esophageal hiatus, a type which has a hernial sac, (2) through the region of fusion of the anlage of the diaphragm, and (3) at sites named under the congenital type in the foregoing paragraph.

*Traumatic Hernia.* Traumatic diaphragmatic hernia may be caused by direct or indirect injury, or by inflammatory necrosis of the diaphragm. In case of indirect injury of the diaphragm, the hernia may occur at any point, including points of embryologic fusion; but the most common sites are the dome and posterior half of the left part of the diaphragm. On the other hand, the injury may occur in the right part of the diaphragm. It usually is the result of a severe, crushing injury. When the hernia occurs through the esophageal opening there is a sac, but when it occurs through the leaf of the diaphragm there usually is no sac. In case of direct injury of the diaphragm, the hernia may occur at any point and is usually the result of penetrating wounds, such as those inflicted by a gun or knife.

Rupture of the diaphragm may be the result of inflammatory necrosis which, in turn, has been caused by subdiaphragmatic abscess, or, again, rupture may follow necrosis caused by drainage tubes which have been introduced into empyematic cavities. In these cases the opening usually is situated in the posterior part of the diaphragm and there is no hernial sac.

In general, the various types of diaphragmatic hernia can be divided clinically into two main classes, depending on the abdominal viscera which are involved. In the first class the stomach is the only abdominal viscus incorporated in the hernia; the herina usually occurs through the esophageal hiatus. In the second class, the intestines with or without the stomach and other abdominal viscera are included in the hernia. Such a hernia usually is of traumatic origin and is caused by laceration of a normal diaphragm. It also

may be of congenital origin and may result from structural deficiency of the diaphragm.

Esophageal hiatus hernia is the most common kind of herniation

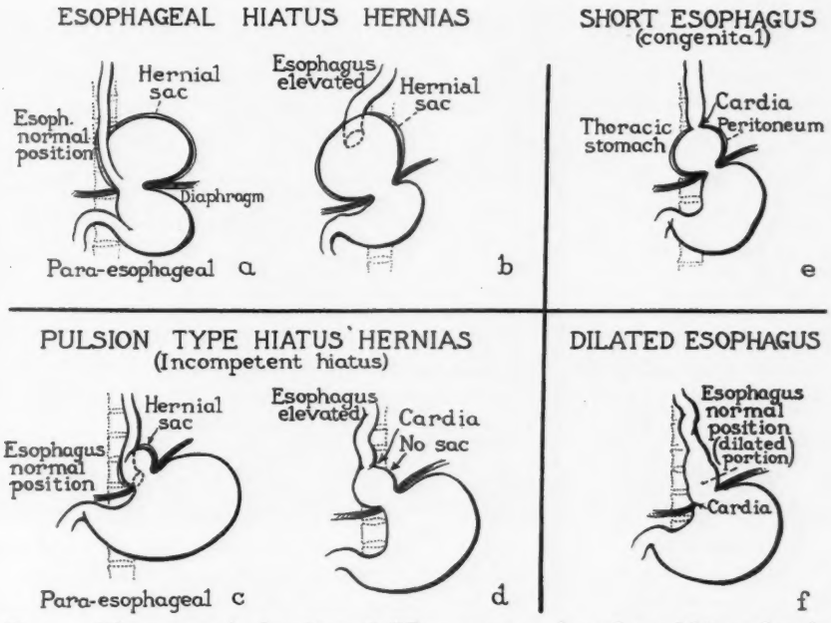


FIG. 10. Diagrammatic drawings of different types of esophageal hiatus hernia and some of the conditions which may simulate hiatus hernias roentgenologically. *a*, esophageal hiatus hernia with herniation of the cardiac end of the stomach through the esophageal hiatus. Esophagus in normal position (para-esophageal hernia). *b*, esophageal hiatus hernia in which there is herniation of the greater portion of the stomach through an enlarged esophageal hiatus into the posterior mediastinum and associated elevation of the esophagus. *c*, slight protrusion of the cardia of the stomach through the hiatus with slight elevation of the lower part of the esophagus to the upper margin of the esophageal hiatus. Pulsion type hernia. Incompetent hiatus. *d*, protrusion of cardiac end of the stomach through the esophageal hiatus and marked elevation of the esophagus into the posterior mediastinum. Incompetent hiatus. *e*, congenital, short esophagus with partial thoracic stomach. Not a true hernia. *f*, dilatation of the lower end of the esophagus, cardiac antrum. Not a hiatus hernia.

occurring through the diaphragm that is found among adult persons. This type of hernia is slowly progressive and constitutes a sliding herniation of the stomach into the posterior part of the mediastinum. It may push into either or both sides of the thoracic cavity, but does not enter the pleural cavity. The stomach usually is the only abdominal viscus involved in the hernia. This type of hernia may progress until the entire stomach is contained within the hernial

sac, and the colon, omentum, and occasionally the spleen may also be drawn into the hernial sac.

I believe it is best to classify all true hernias through the esopha-

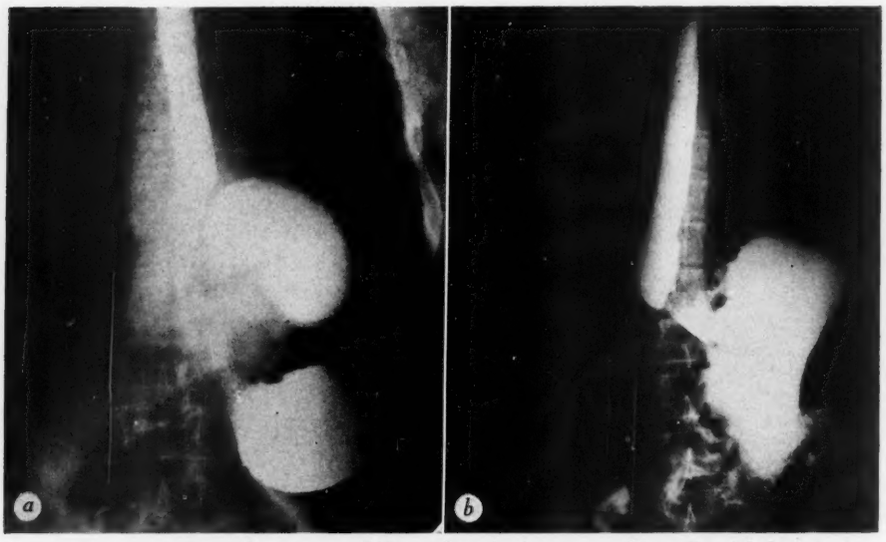

FIG. 11. *a*, patient aged 52 years. Hiatus hernia (para-esophageal) with herniation of cardiac half of the stomach above diaphragm. Esophagus compressed, normal length. *b*, same patient after repair of hernia. Stomach in normal position below diaphragm.

geal hiatus under the one term "esophageal hiatus diaphragmatic hernia." (Fig. 10.) True esophageal hiatus hernias can be divided into two types: In the first type of esophageal hiatus hernia (Fig. 10*a*) the esophagus is of normal length and its lower end is not elevated above the diaphragm, but a portion of the stomach has herniated into the posterior mediastinum, beside the esophagus. This type of hernia is often designated as para-esophageal hiatus hernia. The hernias are usually small to moderate in size and rarely involve more than a fourth to a half of the cardiac end of the stomach, which is contained in the hernial sac. They constitute about a third of the esophageal hiatus hernias on which I have operated. (Figs. 11 and 12.)

In the second type of hiatus hernia (Fig. 10*b*) the esophagus is of normal length but its lower end is elevated above the level of the diaphragm, and the herniated stomach is in the posterior mediastinum. The hernias are usually larger than those of the first type and a third of the stomach or the entire stomach, with a portion of the omentum and occasionally a portion of the colon, is within the

hernial sac. These hernias usually fill the entire mediastinum and generally project into the left side of the thoracic cavity but they may project into the right side of the thoracic cavity or into both

FIG. 12. *a*, patient aged 67 years. Herniation of the pyloric half of the stomach into the posterior mediastinum. Esophagus in normal position with cardiac end of the stomach in the abdomen (para-esophageal). *b*, same case after repair of the hernia. Entire stomach in normal position below the diaphragm.

sides. Hernias of this type constitute about two-thirds of the esophageal hiatus hernias on which I have operated. (Figs. 13 and 14.)

I believe that the essential difference between these two types is one of degree rather than any fundamental difference in origin. Inasmuch as these hernias are progressive in character and are in reality a sliding type of hernia, hernias of the first type may ultimately develop into those of the second type as more of the stomach becomes included in the hernial sac. In many cases of esophageal hiatus hernias there is dilatation of the lower end of the esophagus. This is usually more marked in the larger types of hiatus hernias.

With the more frequent roentgenologic recognition of small hiatus hernias in the last few years, two additional types (Fig. 10 *c* and *d*) have been recognized which may be considered pulsion types of esophageal hiatus hernias. In these types a small portion of the cardiac end of the stomach projects through the esophageal hiatus. They are probably attributable to atrophy of the elastic fibers of the diaphragmatico-esophageal membrane together with relaxation of the circular muscle surrounding the esophageal opening,

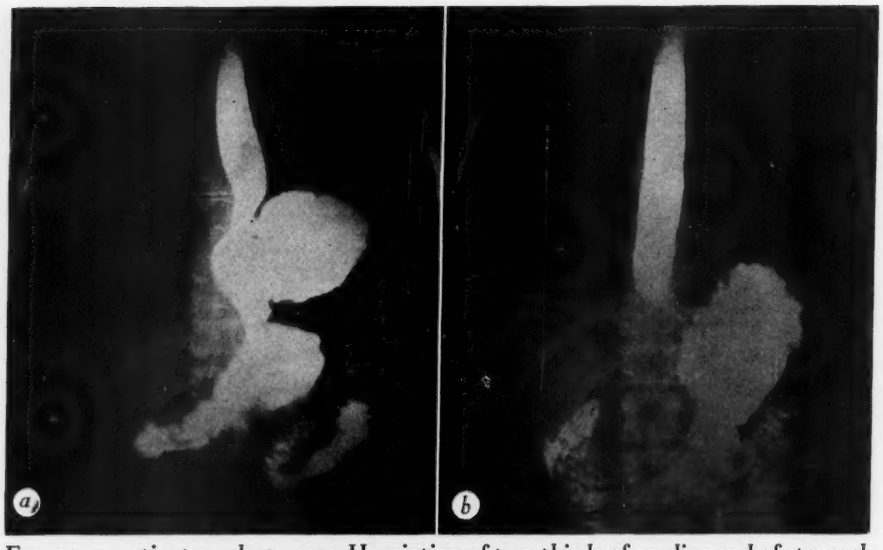

FIG. 13. *a*, patient aged 42 years. Herniation of two-thirds of cardiac end of stomach into posterior mediastinum and extension into left side of thoracic cavity. Marked elevation and displacement of esophagus. *b*, same case after operation. Entire stomach in normal position below elevated left portion of diaphragm. Esophagus normal in length and position.

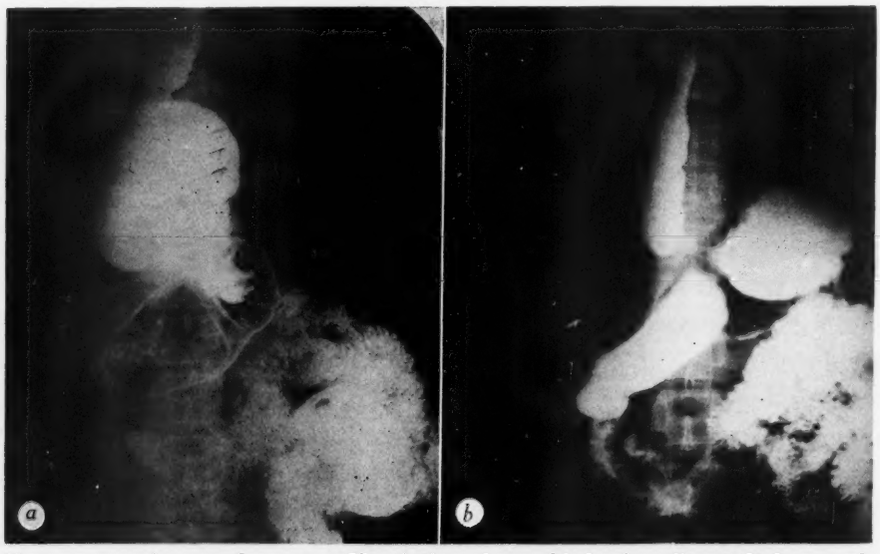

FIG. 14. *a*, patient aged 39 years. Herniation of two-thirds of cardiac end of stomach into posterior mediastinum and extension into right side of thoracic cavity. Marked elevation and displacement of esophagus to right. *b*, same case after operation. Entire stomach in normal position below elevated left portion of diaphragm. Esophagus normal in length and position.

resulting in an incompetent hiatus. (Figs. 15 and 16.) Recognition and differentiation of these two types is often difficult by roentgenologic examination alone. The establishment of the diagnosis is aided by

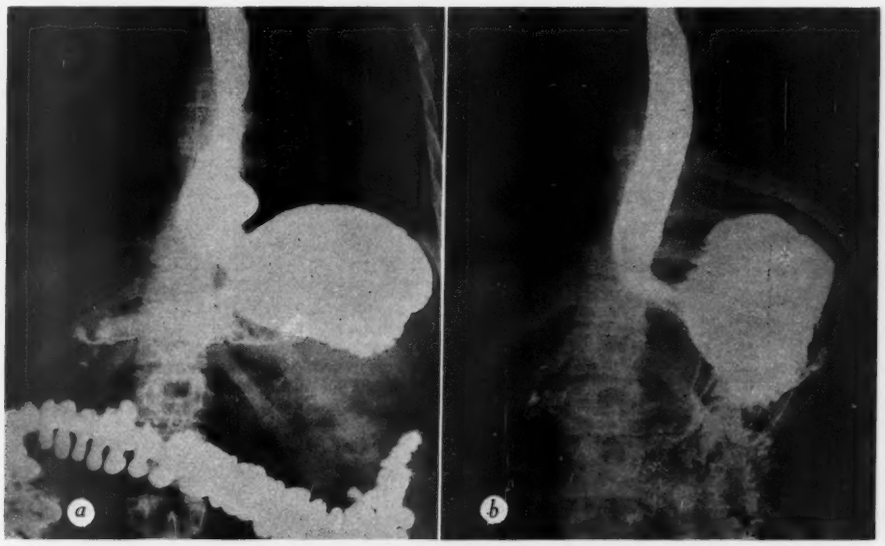

FIG. 15. *a*, patient aged 36 years. Small protrusion of cardiac end of the stomach through esophageal hiatus with distortion of lower part of esophagus. Incompetent hiatus. *b*, same case after operation. Stomach in normal position below the diaphragm and normal contour to esophagus.

an esophagoscopic examination which is often essential in arriving at an accurate diagnosis. These hernias are more commonly seen in elderly people. In many instances the finding of these hernias is incidental as the patients do not present any subjective symptoms; however, in some instances the symptoms may be severe and may not respond to conservative measures. In the latter cases, surgical intervention is necessary to relieve the symptoms.

Hernias of type 3 (Figs. 10*c* and 15) closely simulate the paraesophageal type of hernia except that they are relatively small. There is the beginning formation of a definite hernial sac. The esophagus is only slightly elevated, the abdominal portion being elevated to the superior border of the diaphragm. The hernia may remain small or it may develop into a true paraesophageal hernia.

In the fourth type (Figs. 10*d* and 16) there is incompetency of the entire hiatus with a funnel-like protrusion of the cardiac end of the stomach above the diaphragm and a comparable elevation of the esophagus which enters the apex of the protruded portion of the stomach. This type does not have a true hernial sac and may not be

considered a true hernia. This type of hernia must be distinguished in diagnosis from a congenital short esophagus with partial thoracic stomach and dilatation of the lower end of the esophagus. This is

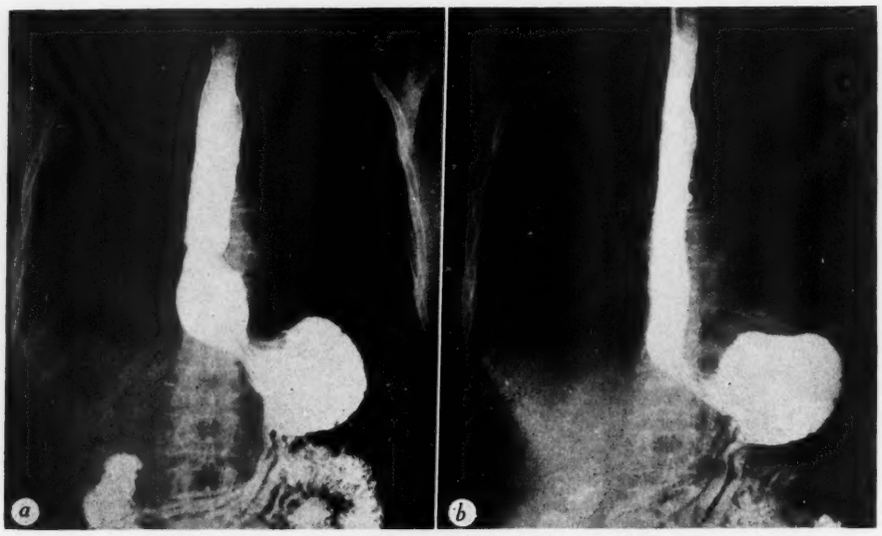

FIG. 16. *a*, patient aged 59 years. Protrusion of the cardiac end of the stomach and elevation of the lower end of the esophagus above the diaphragm. Pulsion type of hernia (incompetent hiatus). *b*, same case after repair. Stomach in normal position below diaphragm. Esophagus normal in length and position.

best accomplished by an esophagoscopic examination to determine the length of the esophagus.

In congenital short esophagus (Fig. 10*e*) because of congenital malformation, the esophagus is not long enough to permit the stomach to reach its normal position below the diaphragm. The stomach is held suspended in the posterior mediastinum above the diaphragm. In this type of case the abnormality is often considered to be an esophageal hiatus hernia, but it is not a true hernia as the stomach has never been below the diaphragm. I believe the condition could be better described as a congenitally short esophagus with partial thoracic stomach. In my experience this condition is relatively rarely found at operation. I have operated on eleven patients who presented all of the roentgenologic signs of short esophagus but at the time of the operation I found that in four cases the esophagus, although its lower end was above the diaphragm, was of sufficient length to reach below the diaphragm when the herniated portion of the stomach was reduced. Of the remaining seven cases, in three there was a slight shortening of the esophagus

associated with a herniation of the cardiac end of the stomach, and the remaining four were cases of true short esophagus with partial thoracic stomach. In all but one it was possible to bring the

FIG. 17. *a*, patient aged 49 years. Congenital short esophagus with partial thoracic stomach; a sixth of cardiac end of stomach suspended above the diaphragm. *b*, same case one month after repair of hernia. Entire stomach in normal position below elevated diaphragm.

stomach entirely below the diaphragm after separation of the attachments around the hiatus and the elevation of the diaphragm resulting from interruption of the phrenic nerve. In one case it was impossible to bring the stomach entirely below the elevated diaphragm and it was necessary to suture a small portion of the cardia into the hiatus when the hiatus was closed. This indicates that it is not always possible by roentgenologic methods to determine the type of hernia present. I believe that this condition, although rare, is an entity but should not be grouped with the true hiatus hernias. (Fig. 17.)

This condition of short esophagus, from an etiologic standpoint, falls into two classes: first, that in which the esophagus is congenitally short, which has been described, and second, that in which ulceration and occasionally malignant neoplasm of the esophagus has caused cicatricial contraction of the lower end of the esophagus. The cicatricial contraction draws the cardia of the stomach above the diaphragm, and the condition usually is associated with stenosis of the lower part of the esophagus.

In considering cases of short esophagus from a surgical standpoint, it is important not to confuse them with true hiatus hernias, for their surgical management is entirely different. In cases of true

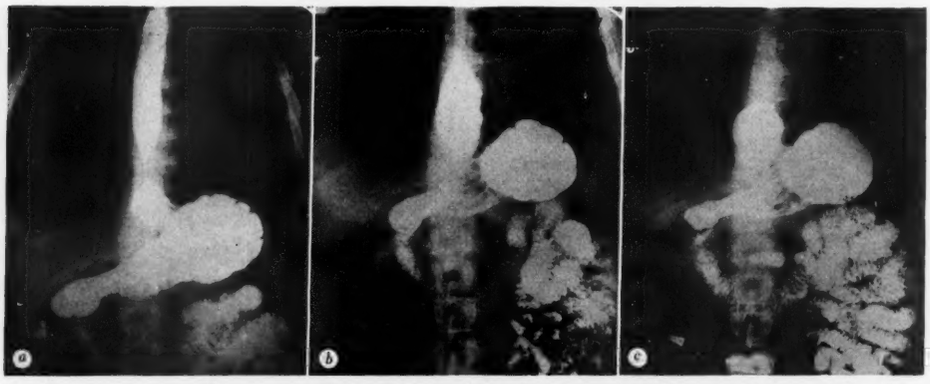

FIG. 18. Patient aged 28 years. Dilatation of the lower end of the esophagus with relaxation of the cardia of the stomach. Not a hiatus hernia. *a*, *b* and *c*, three views taken at the same examination. Operation not performed.

hernia, the surgical problem is that of replacement of the herniated viscera, removal of the hernial sac, and repair of the abnormally enlarged esophageal hiatus. In cases of congenital short esophagus the surgical problem is that of reconstruction and elevation of the diaphragm to a position above the stomach; this can be accomplished if the congenital shortening of the esophagus is not too great. In those cases in which the stomach is elevated above the diaphragm because of traction of cicatrices following ulceration, the symptoms usually are relieved by dilatation of the stenosed esophagus. In those cases due to malignant neoplasms, radical removal of the lesion of the esophagus is to be considered.

*Dilatation of the Lower End of the Esophagus with Relaxation of the Cardia of the Stomach.* (Fig. 10*f*.) This condition not uncommonly simulates esophageal hiatus hernias of the pulsion types both roentgenologically and clinically. In order to establish a definite diagnosis it is usually necessary to examine the esophagus with an esophagoscope as well as to carry out roentgenologic examination. At the time of the esophagoscopic examination, a specimen of tissue can be removed from the wall to determine whether it is squamous cell epithelium of the esophagus or gastric mucosa of the stomach. (Fig. 18.) It is important to differentiate dilatations of the lower part of the esophagus from hiatus hernias as surgical intervention is not indicated in cases of dilatation of the esophagus.

Many writers believe that hiatus hernias are of congenital origin, although some attribute them to certain types of trauma and to senile changes in the tissue of the hiatus. However, because of the relatively few elderly persons who present hiatus hernias roentgenologically, compared to the number of patients whose stomachs are examined roentgenologically, and because of the number of elderly patients whose esophageal hiatuses I have examined in the course of various operations and have not found a hernia present, I believe that the fundamental cause of the hernia, whether the patient is elderly or young, is to be found in the embryologic formation of the diaphragm. The congenitally defective hiatus is not able to withstand the abnormal and, in some instances, the normal pulsion and traction effects of intra-abdominal and intrathoracic pressure. The stress on the hiatus does not have to be great in order to produce hernia. Fewer than 10 per cent of the patients who were proved to have hiatus hernias at operation gave histories of injury. In 50 per cent of these cases some symptoms antedated the injury; this suggests that some degree of herniation had been present prior to the injury.

## INCIDENCE OF DIAPHRAGMATIC HERNIA

The incidence of diaphragmatic hernia is probably no greater now than it was twenty years ago. At that time the condition was thought to be uncommon and its recognition during the life of the patient was rare. A review of the records of the Mayo Clinic revealed that, from January 1, 1908, to and including December 31, 1939, the diagnosis of diaphragmatic hernia had been made either roentgenologically or at operation in 680 cases. The oldest patient was 82 years of age and the youngest patient was 3 months of age. In the first instance of diaphragmatic hernia that was observed at the clinic the hernia was of the esophageal hiatus type and was discovered at operation in 1908. The first roentgenologic diagnosis of a diaphragmatic hernia at the clinic was made in 1911. This hernia was of the traumatic type. The first roentgenologic diagnosis of an esophageal hiatus hernia at the clinic was made in 1921.

The more frequent recognition of diaphragmatic hernia in recent years is exemplified by the fact that a review of the cases in which the condition had been observed at the clinic between January 1, 1908, and December 31, 1925, inclusive, revealed that a diagnosis of this type of hernia had been made in only thirty cases. In fourteen

of the thirty cases the patients were treated surgically. From January 1, 1926 to and including December 31, 1939 on the other hand, about 650 patients had the diagnosis of diaphragmatic hernia. The author operated on 250 of these 650 patients. This study,

TABLE 1

DATA CONCERNING SITUATION, ETIOLOGY AND CONTENTS OF HERNIA

| Site of Opening | Cases No. | Etiology | Cases No. | Contents of Hernia | Cases No. |
|---|---|---|---|---|---|
| Esophageal hiatus | 187 | Congenital (history of trauma, 17) | 187 | Stomach (omentum) | 176 |
| | | | | Stomach, omentum and spleen | 4 |
| | | | | Stomach and colon | 7 |
| Short esophagus type | 11 | Congenital | 11 | Stomach only | 11 |
| Hiatus pleuroperitonealis | 4 | Congenital | 4 | Colon and small bowel | 3 |
| | | | | Colon, small bowel, stomach and spleen | 1 |
| Absent posterior fourth of left side of diaphragm | 4 | Congenital | 4 | Stomach, colon, small bowel and spleen | 3 |
| | | | | Colon and small bowel | 1 |
| Foramen Morgagni (substernal) | 4 | Congenital | 4 | Colon and appendix | 1 |
| | | | | Colon and omentum | 3 |
| Left side of diaphragm | 39 | Trauma: (indirect injury, 28) (direct injury, 7) (inflammatory necrosis, 4) | 39 | Stomach only | 5 |
| | | | | Stomach and colon | 10 |
| | | | | Stomach, colon and small bowel | 7 |
| | | | | Stomach, colon and spleen | 2 |
| | | | | Stomach, colon, spleen and small bowel | 9 |
| | | | | Stomach, colon, small bowel and liver | 4 |
| | | | | Stomach, colon, small bowel, spleen and liver | 2 |
| Right side of diaphragm | 1 | Trauma (indirect injury, 1) | 1 | Stomach, duodenum, colon, small bowel, liver (gallbladder) and head of pancreas | 1 |
| Totals | 250 | | 250 | | 250 |

therefore, disclosed that about twenty times as many diaphragmatic hernias have been recognized in the last fourteen years as had been recognized in the previous eighteen years. I believe that the condition is even more common than the present records indicate. I have examined the diaphragm in the course of other abdominal operations

and occasionally have found a small hernia that had not been recognized either clinically or roentgenologically before operation.

The relative incidence of esophageal hiatus hernia in relation to other types of diaphragmatic hernia, in my experience, is shown by a review of 250 cases of diaphragmatic hernia, in which I operated on the patients from 1926 to 1940. In this series there were 198 cases of esophageal hiatus hernia, four cases of congenital pleuroperitoneal hiatus hernia, four cases in which the hernia was the result of congenital absence of a portion of the left side of the diaphragm, four cases in which herniation occurred through the foramen of Morgagni, and forty cases in which traumatic herniation occurred through the dome of the diaphragm. This shows that herniation through the esophageal hiatus, treated surgically, was between three and four times as common as all other types of herniation combined. (Table 1.)

## SYMPTOMS

The clinical recognition of diaphragmatic hernia on the basis of the subjective symptoms alone is often very difficult. The symptoms are complex because of the various structures involved in the hernia and depend on the amount of mechanical interference with the function of the herniated abdominal viscera, on the degree of impairment of the normal function of the diaphragm and on the amount of increased pressure within the thorax which causes impairment of respiration and circulation.

The clinical syndrome of diaphragmatic hernia may be divided into two main groups: The first group occurs in cases in which the stomach is the only abdominal organ involved in the hernia. These hernias are usually of the esophageal hiatus type. The symptoms are those of intermittent and usually progressive incarceration and obstruction of the stomach. However, this type of hernia may contain various portions of the omentum depending on the amount of stomach involved in the hernia. Inasmuch as these hernias are progressive in character the entire stomach may become involved in the hernia (Fig. 19), and in these hernias the colon may also become incorporated in the hernial sac because of its attachment to the greater curvature of the stomach (Fig. 20); more rarely the spleen may become involved because of its attachment to the cardia of the stomach. These cases involving the colon may present additional symptoms of partial or complete intestinal obstruction.

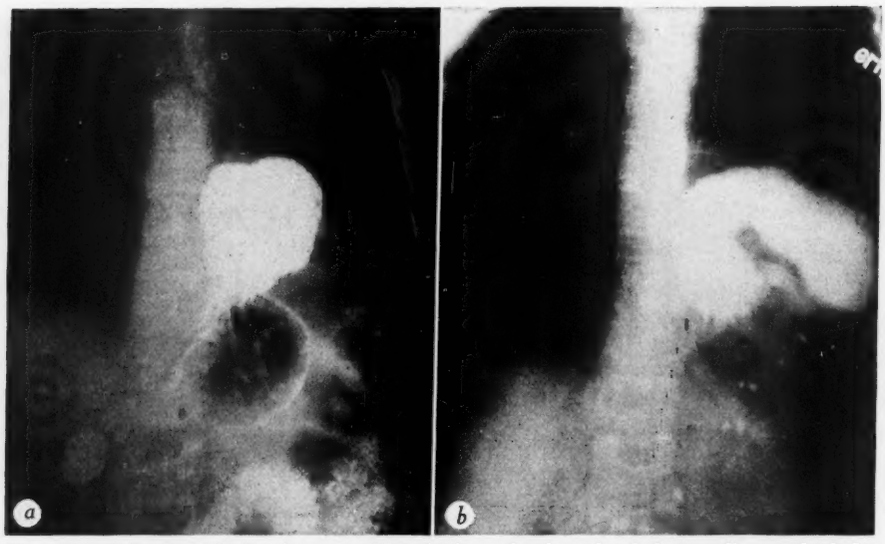

FIG. 19. *a*, first admission in 1930. Herniation of a fourth of the cardiac end of the stomach through the esophageal hiatus into the posterior mediastinum behind the heart. *b*, same case showing progressive development of hernia on second admission in 1935. Herniation of entire stomach through esophageal hiatus into the posterior mediastinum and left side of the thoracic cavity compressing and displacing the esophagus.

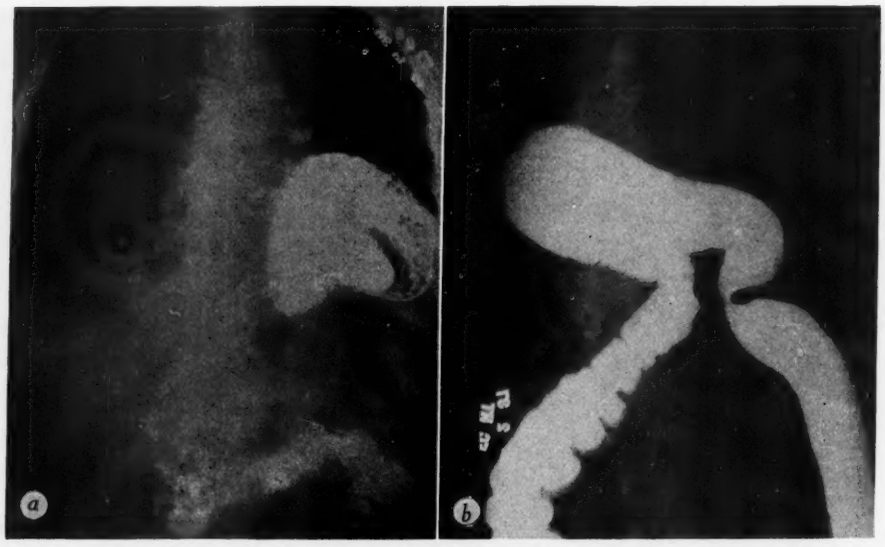

FIG. 20. *a*, herniation of the entire stomach through the esophageal hiatus into the posterior mediastinum. The stomach extends into the left side of the thoracic cavity. *b*, same case. Herniation of the transverse colon through the esophageal hiatus and projecting into the right side of the thoracic cavity.

The second group consists of those cases in which multiple abdominal viscera are involved in the hernia. These hernias are usually of traumatic origin and are caused by laceration of a normal

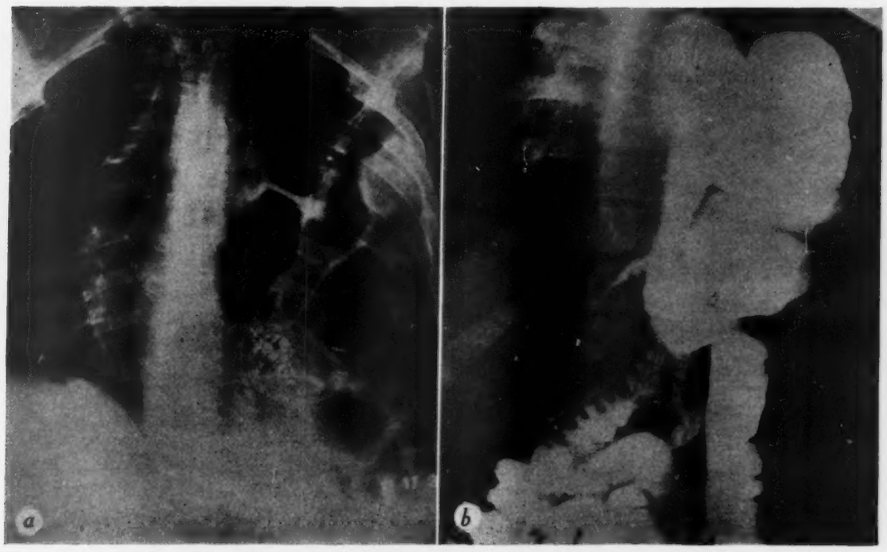

FIG. 21. *a*, entire thoracic cavity completely filled with hollow viscera displacing the mediastinum to the right. Complete collapse of the left lung. *b*, same case. Herniation of the entire stomach and transverse colon into the left side of the thoracic cavity and extending to the apex.

diaphragm. (Fig. 21.) They also may be of congenital origin and may result from congenital structural deficiency of the diaphragm. The symptoms in these cases are more varied and severe in character because of the multiple structures involved and are often more acute in onset. The initial symptom may be that of acute intestinal or gastric obstruction or severe hemorrhage.

*Esophageal Hernia.* Among adults, esophageal hiatus hernia is the most common kind of hernia through the diaphragm. This type of hernia is slowly progressive and constitutes a sliding herniation of the stomach into the posterior mediastinum. It may push into either or both sides of the thoracic cavity but does not enter the pleural cavity because the herniated viscera are confined in the hernial sac, the innermost layer of which is diaphragmatic peritoneum.

The symptoms of esophageal hiatus hernia may begin at birth or at any time during later life. Owing to the progressive character of these hernias the symptoms vary as the hernia becomes larger depending on the degree and type of herniation so that several clinical diagnoses can be made in the same case because of the

changing symptoms. Accordingly, the condition may be termed the "masquerader of the upper abdomen." In a study of 198 cases of this type of hernia in which I have performed operations, it was found that an average of three previous erroneous clinical diagnoses had been made in each of these cases before the correct diagnosis was established. The most common erroneous diagnoses, in order of frequency, were cholecystitis, cholelithiasis, gastric ulcer, duodenal ulcer, hyperacidity, secondary anemia, cardiac disease, carcinoma of the cardia, stricture of the esophagus, appendicitis and intestinal obstruction. In twenty-three of these cases the patients had been operated on previously for other conditions, without complete relief of symptoms and were completely relieved following repair of the hernia. Of these twenty-three patients, thirteen had undergone cholecystectomy previously; four, cholecystostomy; three, appendectomy; two, pyloroplasty and one, gastroenterostomy. (Fig. 22.)

The chief symptoms of esophageal hiatus hernia are pain, distress, gaseous eructation, vomiting, dyspnea, hemorrhage, weakness, anemia and palpitation of the heart. The symptoms of these hernias are more uniform than those of other types of diaphragmatic hernia and often produce a comparatively definite clinical syndrome over a period of many years. No single case will include the entire clinical picture but a study of the symptoms in a large series of these cases has suggested the composite clinical syndrome now to be described.

At the onset the attacks are usually mild; they consist of epigastric distress that is projected through to the back and which comes on in the course of, or shortly after, a heavy meal; but such attacks may be brought on by taking anything into an empty stomach, such as a cupful of coffee. The attacks are usually similar to one another in character but vary a great deal in intensity, depending on the amount of stomach that becomes incorporated in the hernia, the degree of interference with the diaphragm as well as the size of the hernial orifice and the occurrence of associated complications such as traumatic ulcer and incarceration of the stomach. The symptoms are usually relieved by belching of gas and vomiting and are commonly considered to be attributable to cholecystitis (Fig. 22*a*), for which the patient is often treated.

As more of the stomach becomes incorporated in the hernia, the attacks become more severe; the pain is projected straight through to the back and to the lower left side of the thorax, is more marked to the left of the spinal column than elsewhere in the back and often

appears between the scapulae. This pain may be agonizing and difficulty is experienced in belching of gas and vomiting because of spasm of the diaphragm and reflex cardiospasm. The spasm of the

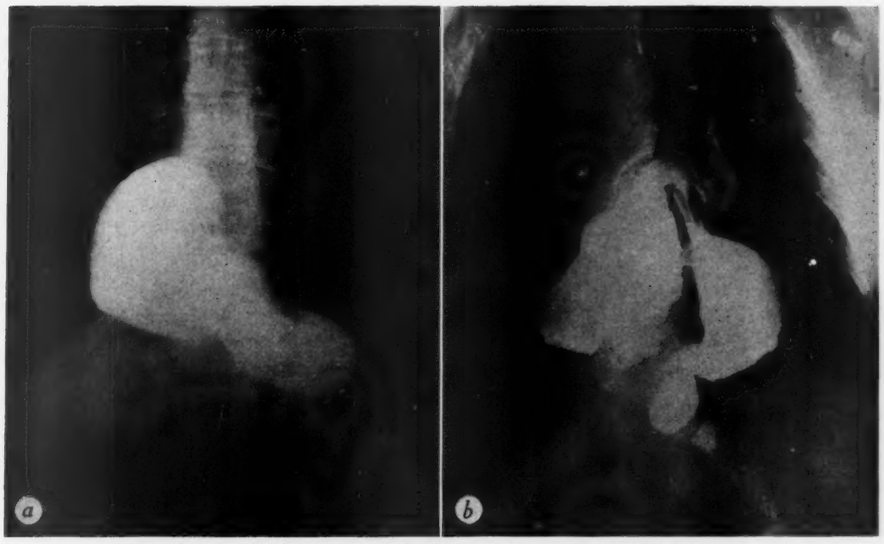

FIG. 22. *a*, herniation of the cardiac fourth of the stomach into the posterior mediastinum extending into the right side of the thoracic cavity. Previously diagnosed and operated on for gallbladder disease. *b*, another case. Herniation of the entire stomach, which is inverted on itself, into the posterior mediastinum. The stomach extends into both sides of the thoracic cavity causing marked pressure on the pericardium. This case previously had been diagnosed as angina pectoris.

diaphragm produces an hourglass deformity of the stomach which interferes with emptying of the upper loculus and causes increased intragastric pressure. The pressure of the herniated portion of the stomach on the lower part of the esophagus also interferes with the belching of gas or vomiting. Spasm of the diaphragm is commonly associated with referred phrenic pain in the left shoulder, which at times may be projected down the arm. The increased pressure within the thorax causes cardiac embarrassment, with palpitation and tachycardia. The pressure on the lung and the interference with the motion of the diaphragm cause dyspnea. These symptoms are augmented when the patient lies down and in the more severe instances of the condition it is necessary for patients to sit up to breathe. The attacks may last for a few minutes to several hours and occasionally they are considered to be caused by coronary sclerosis or by myocardial disease. (Fig. 22*b*.) The attacks usually are com-

pletely relieved by vomiting and often recur immediately after food is taken.

There is often an interval of weeks or months between attacks.

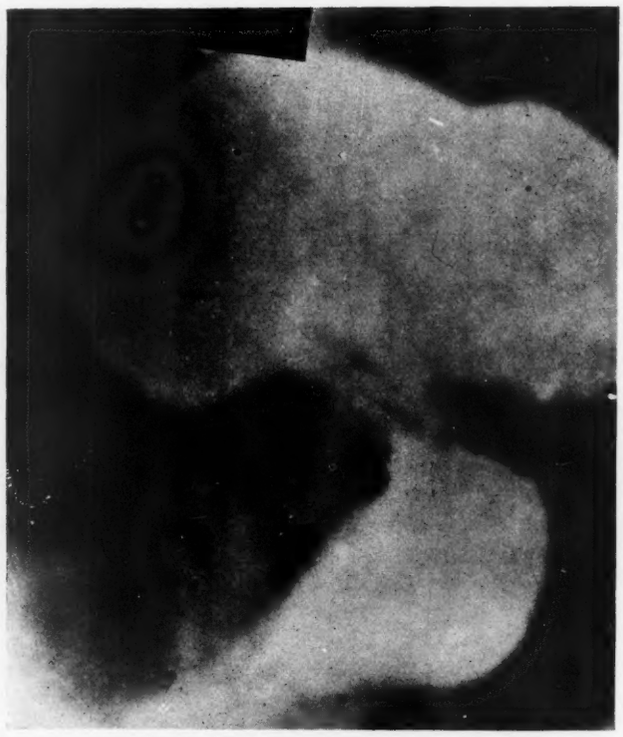

FIG. 23. Herniation of the cardiac three-fourths of the stomach into the posterior mediastinum with marked constriction at the hernial orifice, previously diagnosed and treated as peptic ulcer.

It is probable that during the interval between attacks the stomach is not incorporated in the hernial ring and is situated in its normal position below the diaphragm. When the attacks become more or less constant, the constancy usually indicates that the stomach has become fixed in the thorax by adhesions. All the early symptoms or pressure are augmented during the attacks. There is loss of weight arising from the patient's inability to retain food and from marked restriction in diet resulting from the patient's fear of bringing on an acute attack, which may be termed "food-fear." The vomiting is more severe and often is of the retention type. During the severe vomiting, the vomitus may contain blood. If the attacks are of long standing, the patient not uncommonly has a burning sensation in the

epigastrium after meals, which is relieved by taking small quantities of food. If a large amount of food is taken, it may bring on one of the attacks that is associated with incarceration of the stomach. Many of these patients present a comparatively typical syndrome of peptic ulcer, are given medical care and obtain partial relief because they have taken a restricted amount of food at frequent intervals. (Fig. 23.)

Hemorrhage is not an uncommon sign. It is due to a traumatic ulcer which is usually situated in the lower end of the esophagus close to its juncture with the stomach and it may be found in that portion of the stomach in the hernial sac near the lesser curvature. These traumatic ulcers result from the to-and-fro action of the stomach in the hernial ring when the hernia is small as well as from the forceful pressure exerted on the large distorted and congested stomach during the attacks of vomiting when the hernia is large. There is also the additional factor of regurgitation of gastric juices into the lower part of the esophagus which produces esophagitis.

The bleeding from these traumatic erosions may be severe and hematemesis or melena is often one of the chief signs. This type of hemorrhage is more commonly noted in those cases in which there occur repeated severe attacks of obstruction resulting from incarceration of the stomach in the hernial sac. In some instances the hemorrhage that results from the ulceration in these cases is so severe as to endanger life. These traumatic ulcers may be multiple and are usually relatively superficial and depend on the mechanical derangement of the herniated stomach for their presence. (Fig. 24.) After repair of the hernia and replacement of the stomach into its normal position most of these traumatic ulcerations heal spontaneously. In several instances the traumatic ulcer has become more deeply seated simulating the usual type of peptic ulcer and has not healed after repair of the hernia. In the few cases of this type that I have seen the symptoms have been of long standing or the patient's condition has been one of severe incarceration.

In other instances the patient may not be aware of any bleeding and may have a very marked secondary anemia resulting from occult hemorrhage into the stool. I should like to emphasize the importance of secondary anemia as one of the important clinical manifestations of this type of hernia. It was noted in 11 per cent of the 198 cases in which I have performed operation. However, in all cases in which traumatic ulceration was present secondary anemia

was not present, as in 19 per cent of these cases ulceration was noted on esophagoscopic examination.

The foregoing is the clinical picture of a typical case of slowly

FIG. 24. *a*, herniation of the cardiac end of the stomach through the esophageal hiatus showing defect of the traumatic ulcer at the cardia above the diaphragm. *b*, same case six months after operation showing entire stomach in normal position below the diaphragm. No evidence of traumatic ulcer.

progressive herniation of the stomach through the esophageal hiatus but of course it cannot be expected that in every case all the symptoms will be present, any more than it can be expected that a typical history will be elicited in every case of any other organic condition. The chief symptoms depend on the time in the course of the disease at which the patient is examined, on the rapidity with which the hernia has been produced, on the amount of fixation of the stomach in the thorax and on the amount of disturbed function of thoracic organs.

Patients who have esophageal hiatus hernia, of which the perdominating symptoms are those of esophageal obstruction, are of particular interest and require careful clinical study. The symptoms may be attributable to an entirely unassociated lesion of the lower part of the esophagus, such as cardiospasm, carcinoma, or diverticulum, or they may be the result of ulceration or stricture of the esophagus caused by the hernia. This esophageal ulceration produced by hernia is attributable to the repeated or constant pressure of the hernial sac on the lower part of the esophagus or is caused by

regurgitation of food or gastric secretion into the lower part of the esophagus. The ulceration may be a small, localized lesion or it may involve the entire circumference of the lower part of the esophagus and later may contract and produce a stricture. Because of the possibility of a lesion in the lower end of the esophagus, caused by or unassociated with the hernia, I believe esophagoscopic examination is advisable in all cases. Esophagoscopic examination is also essential in determining definitely the presence of a traumatic ulcer as these lesions are rarely demonstrable by roentgenologic examination.

It is to be remembered that patients presenting symptoms of diaphragmatic hernia may also have symptoms due to other unrelated disease, which confuse the clinical manifestations by presenting a dual complaint. I have operated on several patients who have had an associated duodenal ulcer with obstruction, gastric ulcer with obstruction and carcinoma of the pyloric end of the stomach with obstruction as well as several patients who had cholelithiasis. In cases presenting cholecystic disease as well as an esophageal hiatus hernia, I think it is advisable to operate for the cholecystic disease before the hernia is repaired (unless the hernia is incarcerated or obstruction is present) because of the clinical danger of an acute condition arising from the cholecystic disease that might produce a recurrence of the hernia from severe attacks of vomiting. If a gastric ulcer or a gastric carcinoma which obstructs the pyloric end of the stomach is associated with esophageal hiatus hernia, operation should be performed for both of these conditions at the same time.

*Congenital Hernia.* The symptoms of congenital types of diaphragmatic hernia due to structural deficiency in the formation of the diaphragm usually involve multiple abdominal viscera and are often similar to those noted in association with the traumatic types of hernia as there is rarely a confining sac and the herniated abdominal viscera are in direct contact with the thoracic viscera. The symptoms in these cases are often more severe than those noted in cases of traumatic hernia. Because of the occurrence of the hernia at birth, the respiratory and cardiac symptoms are usually the most severe owing to the marked unilateral alteration in intrathoracic pressure and the occurrence of this derangement of intrathoracic pressure at a time in which the compensatory respiratory and cardiac reserve has not been developed to a sufficient degree to maintain function of these organs. Many infants born with these congenital

defects die in the first few hours or days of life. However, if the respiratory and cardiac mechanisms are able to compensate for the presence of these abdominal viscera in the thorax, these patients may live on to childhood or even adult life without any great amount of disability or symptoms provided that intestinal or gastric obstruction does not develop. There is less likelihood of obstruction developing in these cases than in the cases of traumatic hernia because there are usually less adhesions between the abdominal viscera and the thoracic viscera in the former. When the stomach is involved in these hernias, it usually becomes markedly dilated and these patients often have symptoms of partial gastric obstruction. Intestinal obstruction may occur owing to bands of adhesions between the omentum and loops of bowel or owing to inflammatory conditions of the bowel or appendicitis.

*Traumatic Hernia.* In cases of traumatic hernia the symptoms progress very rapidly, are severe in character, and are attributable to the mechanical interference with the function of the herniated viscera as well as to marked interference with function of the heart and lungs. This is due to the fact that there is no hernial sac and the abdominal viscera are in direct contact with the thoracic viscera. The condition in these cases may be more properly termed "evisceration of the abdominal organs into the pleural cavity" rather than a "true hernia." The most marked immediate symptoms are usually those of respiratory and circulatory embarrassment. These hernias are more frequent in adult life and the compensatory cardiac and respiratory reserve usually carries the patient over the acute symptoms if the other associated injuries have not been too great. Later, severe hemorrhage from the gastrointestinal tract may occur as a result of incarceration or strangulation of the hollow viscera. If the patient survives the acute condition, the later symptoms depend on the viscera involved. The symptoms may consist of obstinate constipation, the occurrence of large quantities of gas in the colon, and attacks of partial or complete intestinal or gastric obstruction. The sudden onset of symptoms in cases of traumatic hernia usually is related directly to the injury and there is rarely a question as to the clinical diagnosis. Surgical treatment is demanded because of the danger of cardiac and respiratory failure or because of intestinal strangulation.

More frequent recognition of the condition in the last two decades has been attributable primarily to development of roent-

genologic methods of diagnosis. Clinical study of proved instances of the condition has established a fairly definite symptomatology which has enabled the clinican to diagnose the condition or to suspect the presence of a hernia and to have a special roentgenologic examination made. It will scarcely be claimed that the entire credit for the present more frequent recognition of this condition is due the roentgenologist, but he is entitled to share this credit with the clinician.

### ROENTGENOLOGIC MANIFESTATIONS

Roentgenography plays an important role in the recognition and diagnosis of diaphragmatic hernia. It is also of great value in determining the size and situation of the defect in the diaphragm, considerations which are of aid in determining the method of surgical treatment is to be instituted.

Larger types of diaphragmatic hernias, and especially those in which a large segment of the stomach or bowel is fixed or incarcerated into the thoracic cavity, are strikingly manifest at roentgenologic examination, and often the diagnosis is self evident. But frequently despite pronounced alteration of the thoracic picture, the diagnosis cannot be established without critical study, and small or reducible hernias are likely to escape discovery unless the examiner is alert for clues that will stimulate thorough search.

Among signs suggestive of hernia that may be elicited during the routine examination of the stomach, displacement of the lower segment of the esophagus is particularly significant and is of common occurrence. In many cases, as the bariumized mixture passes down the gullet, it becomes evident that the lower portion of the esophagus is displaced mesially and that it describes a hooklike curve. In other instances the terminal segment is tortuous but not dilated. In still other cases the segment is angulated. Shortening of the esophagus is noted in the rare instances of congenital shortening. Undue retardation of the barium stream at the hiatus is another potential index of hernia and occurs in many cases.

Scarcely second in importance among signs suggestive of hernia is the observation that the level of the gastric contents is above that of the esophageal aperture. Diaphragmatic hernia is the most probable cause of this condition, and this sign aids in distinguishing hernia from eventration, for in the latter condition the two levels coincide. What apparently is high hourglass contraction of the

stomach with a visible niche at the site of constriction is, in fact, often hernia of the stomach through the diaphragm, and the ulcer is merely a complication. In many cases of hernia the symptoms are chiefly or solely thoracic and roentgenologic examination of the thorax is demanded first. Here again hernias involving the stomach and colon may be obvious, but in many cases the manifestations, although pronounced, are not diagnostic. In such cases hernia always should be taken into consideration and appropriate examination should be requested.

Reducible hernias, such as the para-esophageal variety, usually can be revealed at examination with the patient in the vertical position by pressing the stomach upward or they may be suspected by the presence of one or more of the signs already described, but fluoroscopic examination in the horizontal position is always necessary to confirm the diagnosis and to determine the extent of herniation. In no instance can hernia of the colon be excluded confidently without employing the barium enema, and when only the small bowel is implicated, repeated observations after the patient has ingested an opaque meal are necessary for diagnosis. In the rare cases of hernia through the right arch of the diaphragm, a portion of the liver usually projects through the breach and is likely to be mistaken for a neoplasm. On the whole, however, few diaphragmatic hernias should elude roentgenologic disclosure and specific diagnosis.

Roentgenography not only plays a very important role in the recognition of diaphragmatic hernia but is of equal value in the differential diagnosis of this from other conditions in which the clinical symptoms often simulate those of diaphragmatic hernia. It is also of value in determining the presence of any obstructing lesion of the esophagus. However, these lesions are more accurately determined by examination of the esophagus. I believe it is essential that all patients with diaphragmatic hernia should not only complete roentgenographic examination but also thorough esophagoscopic examination before surgical intervention.

Stricture of the lower part of the esophagus associated with esophageal hiatus types of diaphragmatic hernia is not uncommon; these strictures often result from cicatricial contraction of a traumatic ulcer caused by the hernia. It is important that these associated strictures are recognized before surgical treatment is considered for the hernia because in many instances dilatation of the esophagus will

relieve the symptoms sufficiently so that no surgical treatment is necessary for the hernia. In other instances the esophagus may be shortened so that the hernia could not be reduced by surgical procedures. (Fig. 25*a* and *b*.)

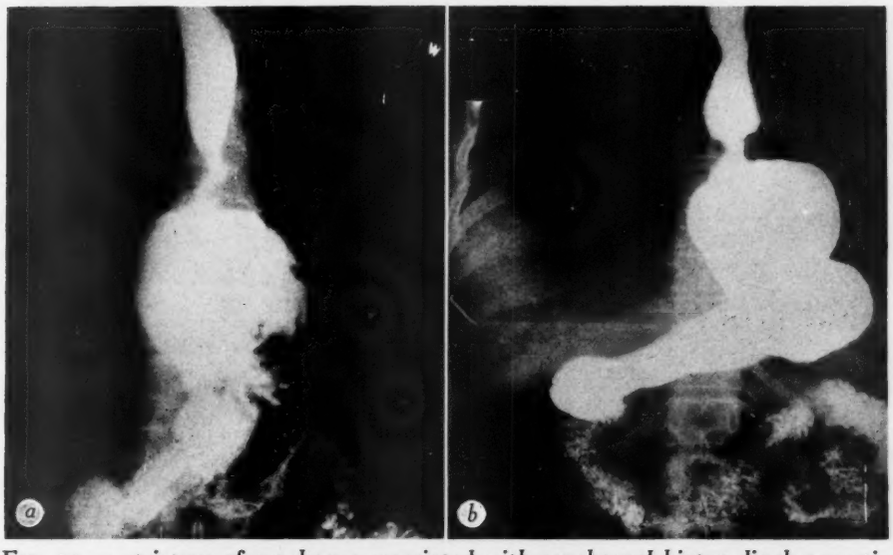


FIG. 25. *a*, stricture of esophagus associated with esophageal hiatus diaphragmatic hernia showing marked contraction of the lower end of the esophagus just above the herniated portion of the stomach. *b*, herniation of the cardiac half of the stomach through the esophageal hiatus with a moderate contraction of the esophagus immediately above the herniated portion of the stomach. Esophagoscopic examination showed the esophageal defect to be due to carcinoma which was proved by tissue removed for microscopic examination.

Carcinoma of the lower end of the esophagus may be associated with diaphragmatic hernia. The presence of these lesions can usually be determined by roentgenographic examination but should be confirmed by esophagoscopic examination with removal of tissue.

Other conditions which are not definitely associated with a hernia may produce clinical symptoms which suggest diaphragmatic hernia and can be distinguished only by roentgenographic or esophagoscopic examination. Some of the more common of these conditions are cardiospasm, diverticulum of the lower end of the esophagus, diverticulum of the stomach close to the cardiac orifice and eventration of the diaphragm. (Figs. 26 and 27.)

## SURGICAL TREATMENT

Diaphragmatic hernia is primarily a mechanical condition, and the only treatment which will relieve the condition is operative

repair or reconstruction of the abnormal opening in the diaphragm. The indications for surgical intervention and the methods and technic of surgical procedures depend on the type, situation and

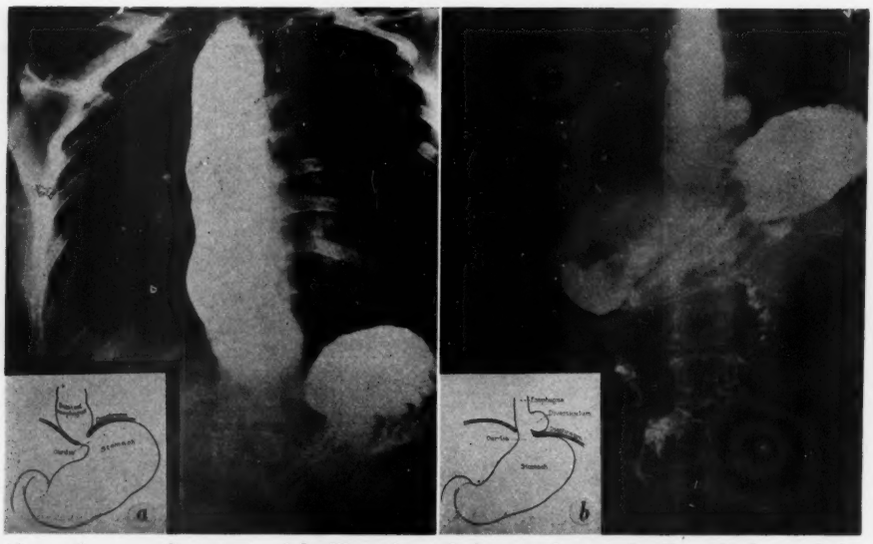

FIG. 26. *a*, cardiospasm with dilatation of the esophagus. *b*, diverticulum of the lower end of the esophagus.

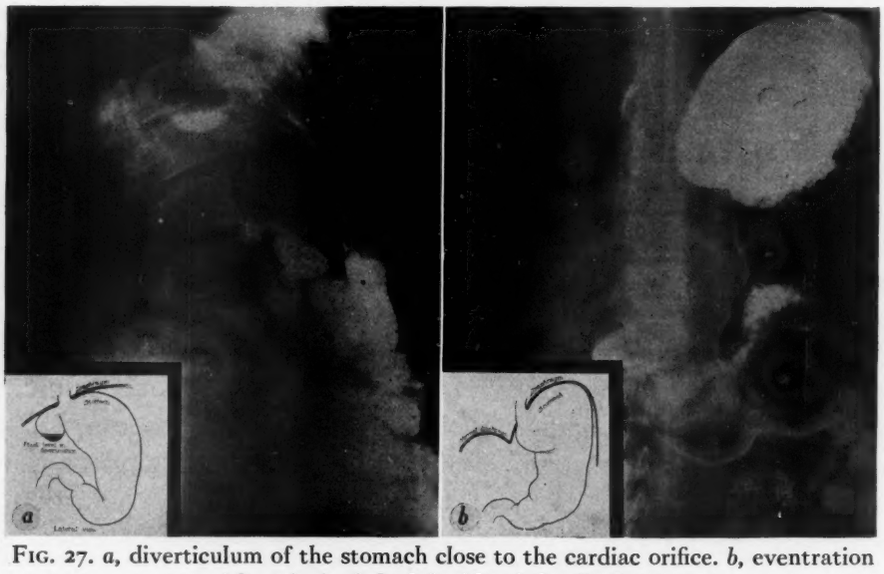

FIG. 27. *a*, diverticulum of the stomach close to the cardiac orifice. *b*, eventration (elevation) of the left side of the diaphragm.

size of the defect in the structure of the diaphragmatic muscle (Fig. 28), the kind and amount of abdominal viscera involved in the hernia, and whether or not the viscera are enclosed in the

hernial sac. I shall first describe the general surgical methods to be used and then consider the special technic which is required in the surgical treatment of some types of hernia.

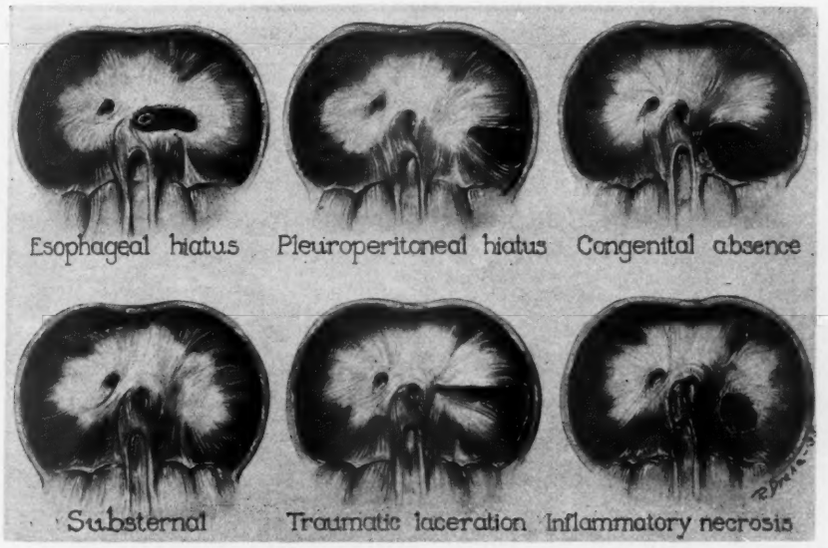


FIG. 28. Situations of congenital structural defects and traumatic lacerations of the diaphragm which cause the more common types of diaphragmatic hernia. (*West. J. Surg.*, 44: 255, 1936.)

From the standpoint of treatment cases of hiatus hernia may be divided into three groups: In the first group the hernia is small and is recognized roentgenologically, often during the course of a general examination, and causes few or no clinical symptoms. No treatment is indicated in this group of cases. The second group includes those cases in which the symptoms are moderate and the hernias are of moderate size; in many of the cases in this group, conservative treatment, such as regulation of diet and reduction of weight, is sufficient to relieve the symptoms. The third group includes those cases in which there is no response to conservative measures; in these cases the hernias usually are large, and in many cases, in my experience, there are complications, such as incarceration of the stomach or gastric erosion. In this group of cases the only treatment that assures relief of symptoms is operative repair of the hernia.

In all cases in which a third or more of the stomach is involved in the hernia, surgical intervention should be considered because the condition is progressive and usually the progressive enlargement becomes more rapid after the hernia has attained this size. Operation should be performed before severe incarceration, with consequent

obstruction and traumatic lesions of the stomach, has occurred. The operative risk is increased by gastric retention, and the technical difficulties are enhanced by fixation of the stomach to the diaphragm and to the hernial sac within the thorax. In all cases in which the colon is involved in the hernia, early operation is necessary because of the danger of occurrence of intestinal obstruction.

Other types of hernia, such as traumatic hernia or those in which there is a congenital absence of a portion of the diaphragm, should be treated surgically, because the colon and small bowel are usually involved in the hernia and there is great danger of the occurrence of intestinal obstruction. In cases of traumatic hernia it is best not to operate until the acute symptoms caused by the primary injury have subsided, if the patient's condition will permit this delay.

*Interruption of the Phrenic Nerve.* Paralysis of the diaphragm, produced by either temporary or permanent interruption of the phrenic nerve, is of value as a procedure performed preliminary to radical operative repair of many different types of diaphragmatic hernia. It is a necessary procedure in the surgical treatment of partial thoracic stomach resulting from a congenitally short esophagus. In some cases in which radical operative repair is contraindicated, it may be used as a palliative measure. In most instances in which interruption of the phrenic nerve is utilized as a procedure preliminary to radical operative repair of the hernia, I prefer, first, to perform temporary interruption of the nerve by crushing it in the cervical region because in many instances it may not be necessary for the resultant paralysis to be permanent. Function is usually reestablished within three to six months. In cases in which reestablishment of function of the diaphragm is not desirable because of the danger of recurrence of the hernia, the paralysis can be made permanent by cutting or avulsing the phrenic nerve. As a procedure preliminary to radical surgical treatment, interruption of the phrenic nerve often is of value in the treatment of incarcerated and strangulated hernias because it prevents spasm of muscle and causes relaxation of the hernial ring. It is of great advantage in the closure of large hernial openings when there is considerable loss of structure of the diaphragm, as is usually found in traumatic or congenital hernias.

In this type of hernia interruption of the phrenic nerve may be done in the cervical region preliminary to the abdominal repair of the hernia or the nerve may be crushed or cut along its course in the

mediastinum through the hernial orifice at the time of operation. The relaxation of the diaphragm following this procedure permits the structural defect to be closed without tension; and in cases of traumatic hernia in which the diaphragm has been torn from the thoracic wall, it permits the diaphragm to be sutured to the intercostal muscles.

Interruption of the phrenic nerve may be utilized as a palliative measure in esophageal hiatus diaphragmatic hernia when the radical operative procedure of closure of the enlarged esophageal hiatus is contraindicated because of the patient's condition and when the stomach is the only abdominal viscus involved in the hernia. The purpose of this procedure is to prevent spasm of the diaphragm, which is the cause of the severe instances of incarceration of the stomach in the hernial sac.

Phrenic nerve interruption without operative repair of the hernia does not completely relieve the symptoms. There is always a moderate amount of gastric distress immediately after, or shortly after, the ingestion of heavy meals, but the patients maintain themselves rather well if they are careful with their diet. This procedure is not applicable to hernias in which a large portion of the stomach lies in the thorax, causing marked pressure on the heart and lungs, nor is it applicable in any case in which the intestines are involved in the hernia. This procedure should not be employed when radical operative repair can be effected.

*Radical Surgical Repair. Anesthesia.* The method of administration of the anesthetic agent depends on the type of hernia which is present. In all cases in which there is no hernial sac, and in which there is a direct communication between the abdominal and thoracic cavities, I prefer intratracheal administration of the anesthetic agent by means of a positive pressure machine. The anesthetic agent to be used depends on the indications presented in each individual case. I have used ether, ethylene and cyclopropane. I prefer the latter. In cases in which there is a hernial sac, as in the esophageal hiatus type of hernia, the anesthetic agent can be administered by the closed mask method.

*General Technical Considerations.* In the treatment of all hernias that have occurred through the left portion of the diaphragm, I prefer the abdominal approach by means of an oblique left rectus incision, starting at the ensiform cartilage and extending to the outer border of the rectus muscle. I believe there is less risk of the

occurrence of thoracic complications when this approach is used. It is of particular advantage in cases of esophageal hernia, for the herniated stomach is usually confined in a sac in the posterior part of the mediastinum and does not enter the true pleural cavity.

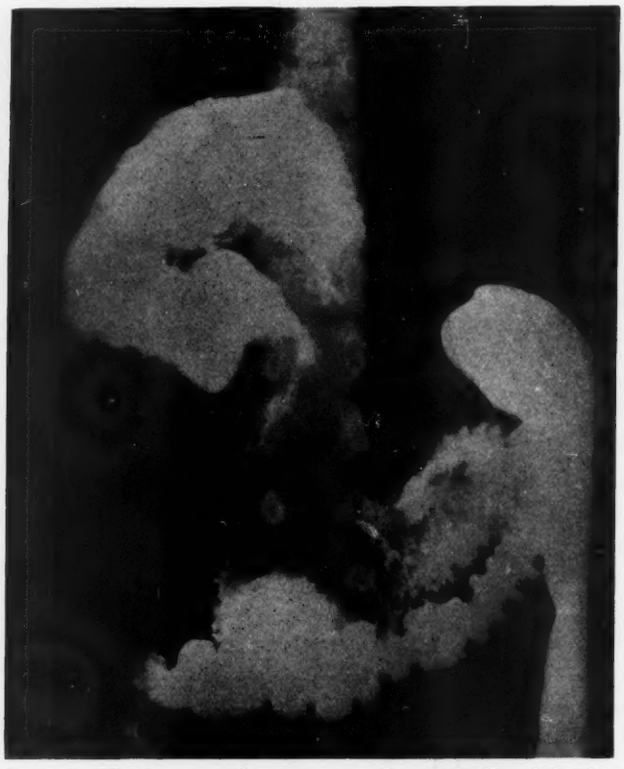

FIG. 29. Patient aged 11 years. Congenitally defective esophageal hiatus with herniation of entire stomach and part of duodenum into mediastinum and right side of thoracic cavity.

In the repair of hernias through the right portion of the diaphragm, I prefer the thoracic approach because the large, right lobe of the liver makes the abnormal opening in the diaphragm inaccessible from the abdominal approach.

The technical difficulties of adequate exposure of the hernial openings through the left portion of the diaphragm and the esophageal hiatus are often considerable because of fixation of the left lobe of the liver to the leaf of the diaphragm. The exposure of these hernial openings is greatly facilitated by cutting the suspensory ligament and retracting the left lobe of the liver to the right. This can be accomplished, when the left lobe is small, by folding it on

itself, and when it is large, by retracting it forward into the wound. The spleen is often very adherent to the posterior part of the diaphragm and hernial openings, but usually can be separated from

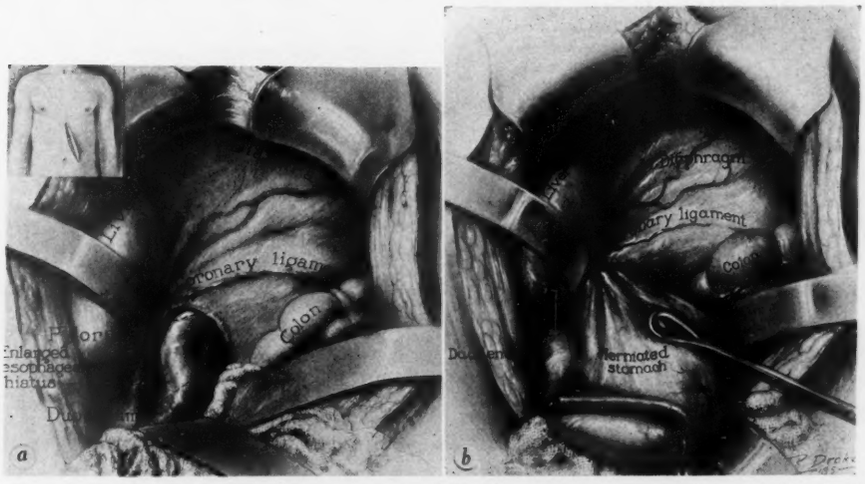


FIG. 30. *a*, same case as that represented in Figure 29, at operation. Abdominal approach. Duodenum protruding from the structurally defective and enlarged esophageal hiatus. *b*, removal of herniated stomach from hernial sac in mediastinum and right side of thoracic cavity.

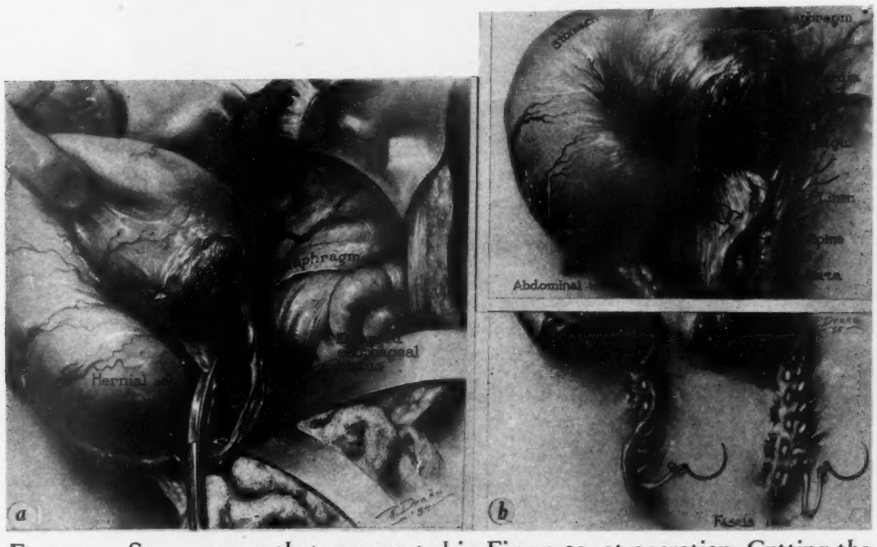


FIG. 31. *a*, Same case as that represented in Figure 29, at operation. Cutting the hernial sac from its attachment to the stomach. This sac was left in the posterior mediastinum. *b*, Closing the structurally defective esophageal hiatus by overlapping margins. Tissue reinforced with fascia lata.

these structures by blunt dissection. In some instances the spleen has been so traumatized by the injury, and so bound into its abnor-

mal position by adhesions, that it cannot be separated from the hernial opening without seriously injuring it. This not uncommonly occurs in the traumatic types of hernia, and occasionally in esophageal hiatus hernias. In these cases splenectomy is necessary.

FIG. 32. Same case as that represented in Figure 29, after operation. Stomach in normal position below repaired left side of diaphragm. Right lung fully expanded.

*Esophageal Hiatus Hernia.* Hernias through the esophageal hiatus are true hernias and have a hernial sac. (Figs. 29, 30, 31 and 32.) The attachment of the sac to the stomach must be separated and the sac must be either completely removed or permitted to retract into the posterior portion of the mediastinum. I believe that this is one of the most important technical considerations in the surgical treatment of these hernias.

After the sac has been removed, the enlarged esophageal hiatus is repaired by overlapping the margins of the opening. Closure is usually made to the left of the esophagus but in some cases it is necessary to close the opening partially both to the right and left of the esophagus. (Fig. 33.) In a few instances the enlargement of the esophageal opening is posterior, extending to the spinal column

and requiring the overlapping of the margins posterior to the esophagus. In such cases, the condition is often thought to be a herniation through the aortic opening, but extending over the aorta there

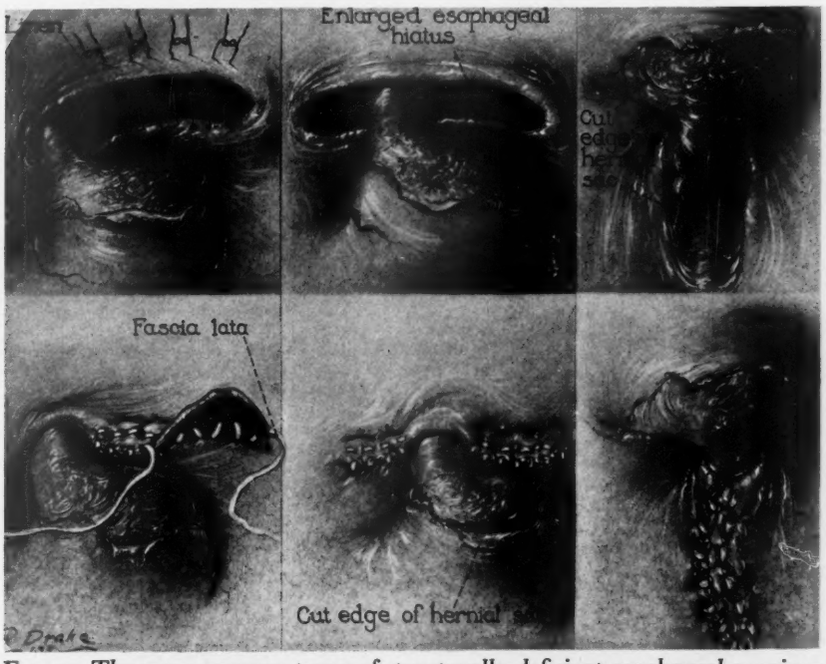


FIG. 33. The more common types of structurally deficient esophageal openings which permit herniation of the stomach into the posterior mediastinum and the method of repair of each type of opening with fascia lata and linen after removal of the hernial sac. (*West. J. Surg.*, 44: 255, 1936.)

usually is an imperfectly developed, fibrous band which is the margin of the defective esophageal hiatus. The closure is usually made with living sutures of fascia lata which are removed from the thigh. (Fig. 34.) The overlapped margins of the hernial opening are first stabilized with interrupted linen sutures. The fascia lata is then woven into the tissues by continuous suture and fixed in the tissues with interrupted linen sutures.

In many cases in which the stomach is incarcerated (Fig. 35) or obstructed, it is impossible to pass a stomach tube into the obstructed loculus of the stomach before operation. In these cases it is advisable to pass a stomach tube soon after the abdomen is opened, directing the tube into the obstructed portion of the stomach in order to empty the gastric contents before any attempt is made to reduce the herniated viscera, because of the danger of regurgitation and aspiration of gastric contents into the lung.

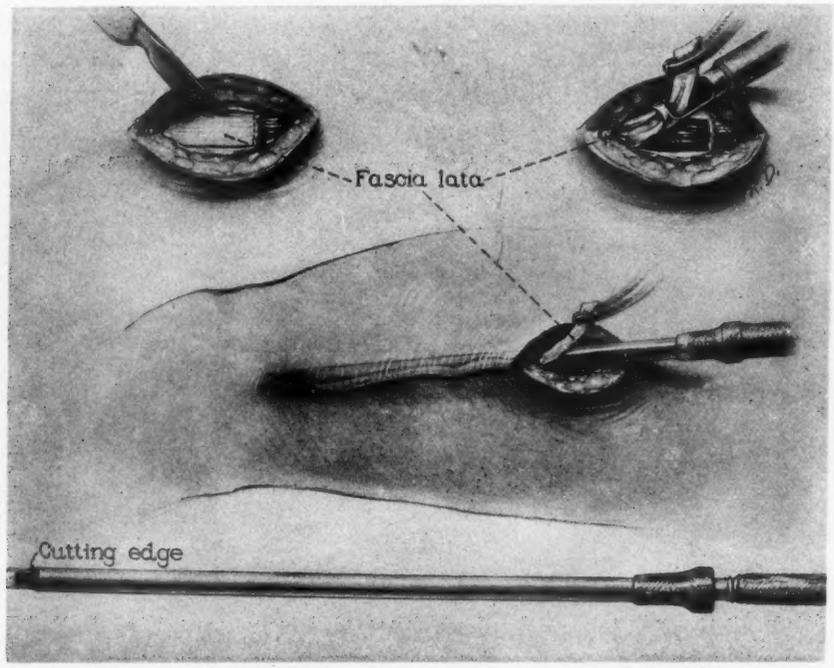


FIG. 34. Method of removal of the fascia lata from the thigh with a Masson fascial stripper. The fascia is divided into strips and utilized in repair of the hernia.

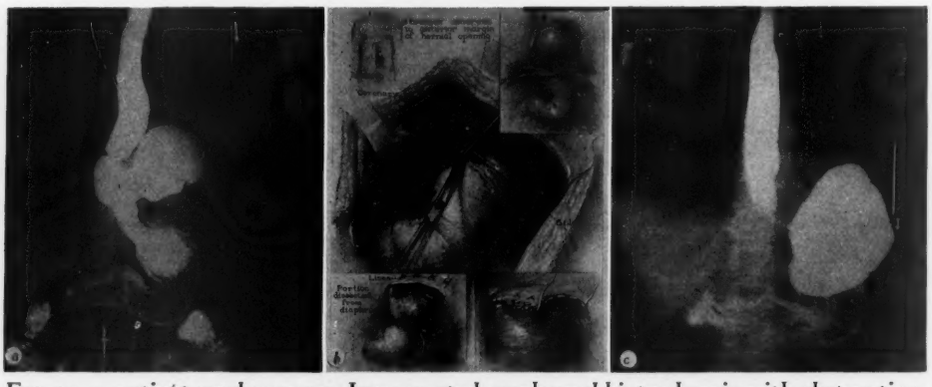

FIG. 35. *a*, patient aged 55 years. Incarcerated esophageal hiatus hernia with obstruction. Herniation of cardiac half of stomach with elevation and displacement of lower part of esophagus. *b*, same case at operation. Separation of adhesions fixing stomach to margins of hernial orifice. Inserts reveal repair of hernia with linen and fascia lata. *c*, same case after repair of hernia. Stomach in normal position below elevated left side of diaphragm. Esophagus in normal position.

Before closure of the defective esophageal hiatus is completed around the lower part of the esophagus, it is important that a stomach tube of large caliber be passed through the esophagus into

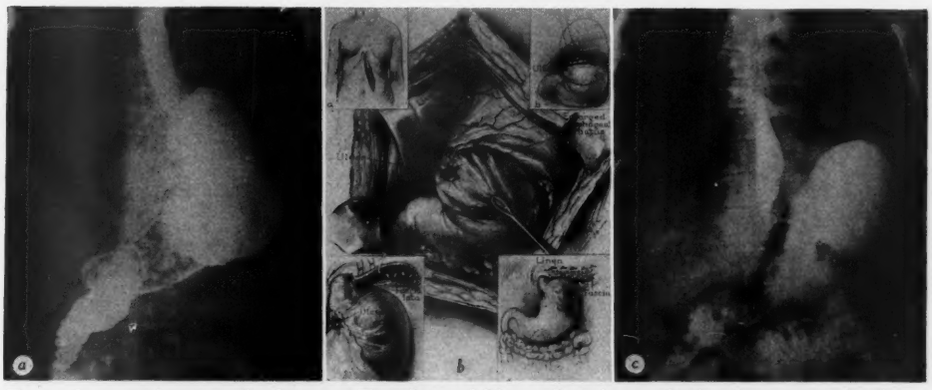

FIG. 36. *a*, patient aged 56 years. Esophageal hiatus hernia producing large gastric ulcer. Acute incarceration of cardiac end of stomach in hernial sac with large gastric ulcer high on lesser curvature. *b*, same case at operation, showing large traumatic ulcer at the junction of the esophagus and cardia of the stomach on the lesser curvature with inflammatory adhesions fixing the stomach to the inner margin of the hernial opening. Inserts reveal repair of esophageal hernia with interrupted sutures of linen and with fascia lata after replacement of the stomach into the abdomen and gastroenterostomy after closure of the traumatic ulcer. *c*, same case after operation. Entire stomach in normal position below elevated left side of diaphragm. Posterior gastroenteric stoma is functioning normally.

FIG. 37. *a*, patient aged 64 years. Esophageal hiatus diaphragmatic hernia with herniation of two-thirds of cardiac end of stomach and upper pole of spleen into posterior mediastinum. *b*, same case at operation. Abdominal exposure of herniated stomach and spleen. Inserts show repair of esophageal hiatus with linen and fascia lata. *c*, same case after repair of hernia and splenectomy. Stomach in normal position below elevated left side of diaphragm.

the stomach, to aid in the reconstruction of the normal esophageal opening and to prevent constriction of the esophagus by a tight

closure. The loose areolar tissue or a small portion of the esophageal wall at the cardia is incorporated in the innermost margin of the closure by a suture of chromic catgut.

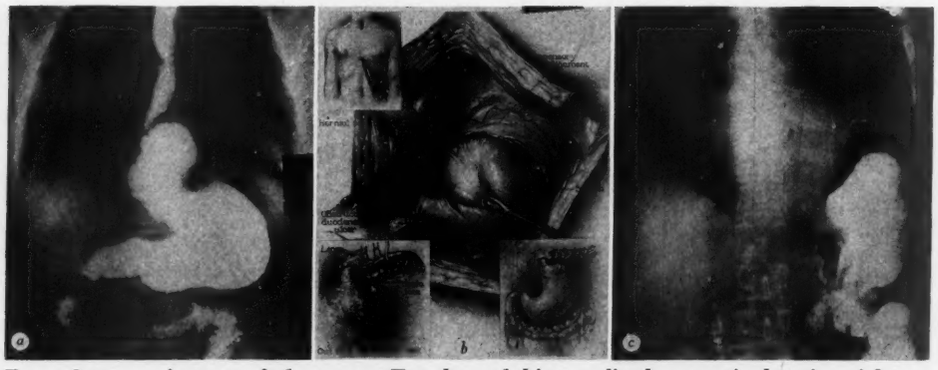

FIG. 38. *a*, patient aged 63 years. Esophageal hiatus diaphragmatic hernia with an obstructing duodenal ulcer. Herniation of cardiac third of the stomach through the esophageal hiatus into posterior mediastinum compressing and displacing lower third of esophagus. *b*, same case at operation. Removing herniated stomach from mediastinum. Large, obstructing, perforating duodenal ulcer. Closure of enlarged esophageal hiatus with fascia lata and posterior gastroenterostomy. *c*, same case after operation. Entire stomach in normal position below left side of diaphragm, elevation of which is due to interruption of phrenic nerve. Gastroenteric stoma free.

Not uncommonly in these cases there is an associated traumatic ulcer in the herniated portion of the stomach, along the lesser curvature, close to the cardia (Fig. 36), and this ulcer is often adherent to the margins of the hernial opening. Great care should be used in replacing the stomach in the abdomen, and in removing the sac from the stomach because of the danger of perforating this thinned-out portion. In cases in which the ulcerated portion is penetrated, it should be repaired immediately with continuous catgut and linen sutures. The spleen is not uncommonly involved in the hernia of the esophageal hiatus type. In a few cases, particularly those in which the opening is posterior, the spleen may not only be adherent to the margin of the opening but may be herniated into the thoracic cavity. In these cases there is usually considerable obstruction to the stomach. The separation of the adhesions in order to replace the stomach and spleen into the abdomen may so traumatize the spleen that its removal may be necessary. (Fig. 37.)

The abdomen always should be thoroughly explored for the presence of any other lesion, particularly of the stomach or gall-bladder. In some cases it may be necessary to operate on other associated lesions. However, I do not believe it advisable to carry out

any additional surgical procedure at the time of repairing the hernia, unless it is imperative, but it is well to know whether the patient has gallstones or any other lesion in the upper part of the abdomen, which might account for subsequent symptoms. (Fig. 38.)

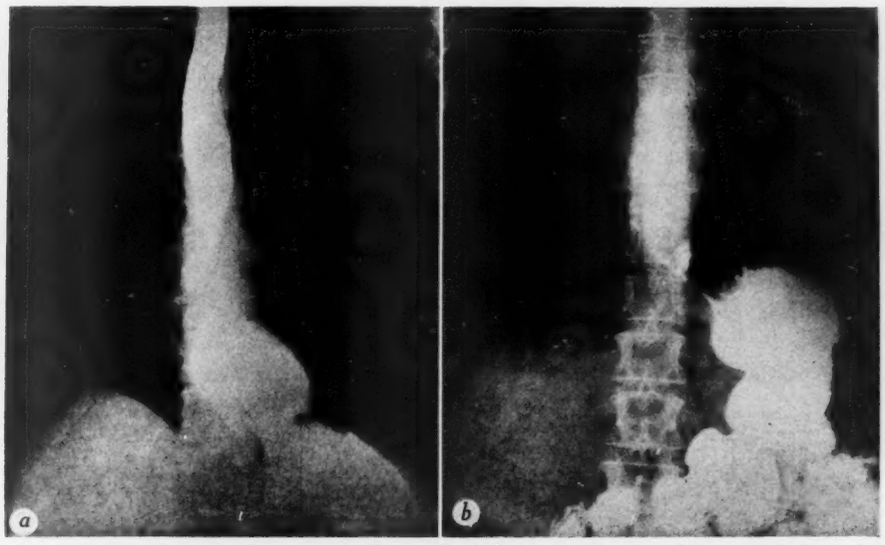

FIG. 39. *a*, patient aged 53 years. Congenital short esophagus with partial thoracic stomach. Approximately a sixth of the stomach is suspended above the diaphragm by the short esophagus which enters the upper part of the stomach. Left side of diaphragm in normal position. *b*, same case after operation. Stomach entirely below elevated left side of diaphragm. Note position of left side of diaphragm as compared to that of right side both before and after operation.

*Congenital Short Esophagus.* The surgical treatment of congenital short esophagus with partial thoracic stomach presents an entirely different technical problem from that of esophageal hiatus diaphragmatic hernia. As pointed out previously, the essential consideration in the surgical treatment of esophageal hiatus hernia is that of replacement of the herniated stomach into the abdomen, the removal or obliteration of the hernial sac and the repair and reconstruction of the esophageal hiatus accurately around the esophagus.

Congenital short esophagus with partial thoracic stomach is not a true hernia of the diaphragm in that the stomach has never been in its normal position below the diaphragm because of shortening of the esophagus. The surgical problem in these cases is that of reconstructing the diaphragm over the elevated portion of the stomach; this can be accomplished if the shortening of the esophagus is not too great. By interruption of the phrenic nerve (complete and

permanent) the diaphragm usually can be elevated from 2 to 5 cm. and then by completely separating the attachment of the esophagus from the attachments around the esophageal hiatus, from 2 to 3 cm. of the esophagus can be drawn down into the abdomen. The elevation of the diaphragm and the pulling down of as much as is possible of the esophagus into the abdomen permit the esophageal hiatus to be closed around the lower end of the esophagus, placing what was formerly the thoracic portion of the stomach below the diaphragm. (Fig. 39.)

*Congenital Diaphragmatic Hernias Due to Malformation and Structural Deficiencies.* These hernias may occur in either the right or the left side of the diaphragm but are much more common through the left side. The more common hernias of this type are those through the pleuroperitoneal hiatus, those due to the lack of formation of the posterior portion of the diaphragm and those through the foramen of Morgagni (Larrey's space), anteriorly, more accurately termed substernal hernias.

In the first two types there is rarely if ever a hernial sac and the abdominal viscera are in direct contact with the thoracic viscera. In the latter type (substernal) there is always a hernial sac which consists of peritoneum and parietal pleura.

The anesthetic agent in the first two types of hernias should be administered under positive pressure after introduction of an intratracheal tube because of the dangers associated with open pneumothorax, as these hernias do not have a hernial sac and are essentially eviscerations of the abdominal contents into the thorax. In the latter type, substernal hernias, the anesthetic agent may be administered with a closed mask.

In the surgical treatment of these hernias the approach in the first two types may be either thoracic or abdominal but I prefer the abdominal approach through an oblique left rectus incision. In the latter type (substernal) the approach should always be through the abdomen and usually through an oblique right rectus incision or a transverse incision in the epigastrium. I prefer the oblique right rectus incision.

*Pleuroperitoneal Hiatus Hernias.* These hernias occur in the posterolateral portion of the diaphragm and are due to failure of fusion of the pleuroperitoneal membrane and the septum transversum. The defect is usually triangular in shape with the apex toward the median portion of the diaphragm. The defect usually

extends to the chest wall but occasionally there is an imperfectly developed band of muscle tissue extending along the chest wall. These hernias do not have a hernial sac and there is a direct communication between the abdominal and the thoracic cavity.

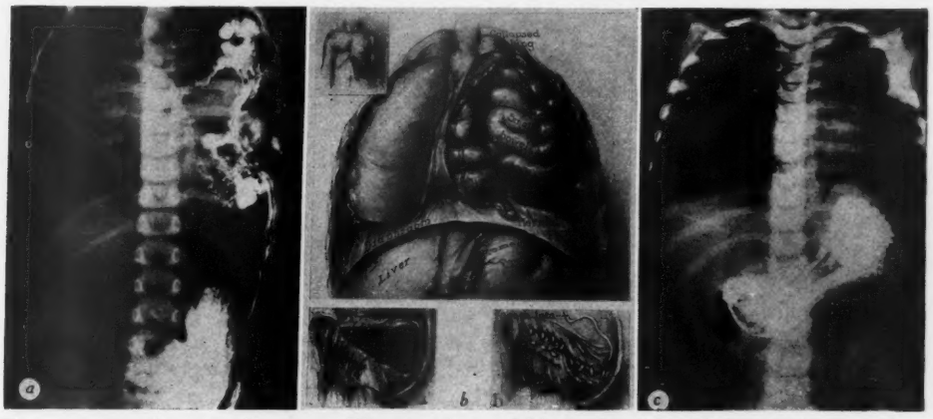


FIG. 40. *a*, patient aged 3 years. Pleuroperitoneal hiatus hernia with herniation of colon and small bowel extending to apex of left side of thoracic cavity, compressing lung and pushing mediastinum to right. Dilated stomach visualized in the lower part of the abdomen. *b*, same case at operation. Left side of thoracic cavity filled with large and small intestine collapsing left lung and pushing mediastinum to right. Insert *a*, persistent pleuroperitoneal hiatus defect. Insert *b*, closure of congenital defect in diaphragm with interrupted silk and continuous fascia lata. *c*, same case on dismissal. Mediastinum in normal position. Left lung fully expanded. Herniated viscera in normal position below diaphragm.

The most common abdominal viscera involved in this type of hernia are the colon and small bowel. There may or may not be herniation of the spleen and stomach. There is often a failure of rotation of the colon and the entire right colon (appendix and cecum) with the terminal part of the ileum and all of the small intestines to the jejunum are involved in the hernia. (Fig. 40.)

This type of hernia is said to be the most common of the congenital types of hernia due to structural deficiencies. These hernias are present at birth. Many of the infants suffering from them die in the first few hours or days of life because of respiratory and cardiac embarrassment and before surgical intervention can be instituted. In treating those infants who are able to survive in spite of the altered intrathoracic pressure and thoracic visceral relationship, surgical intervention should be instituted as soon as possible because of the danger of intestinal obstruction. If they are able to maintain nourishment, it is well to delay operation for two to three months to permit some development of their accessory respiratory mechanism.

If operation is delayed for a long period, the abdominal viscera will have lost their right of residence in the abdomen in that the abdominal cavity will not have developed sufficiently to contain them and

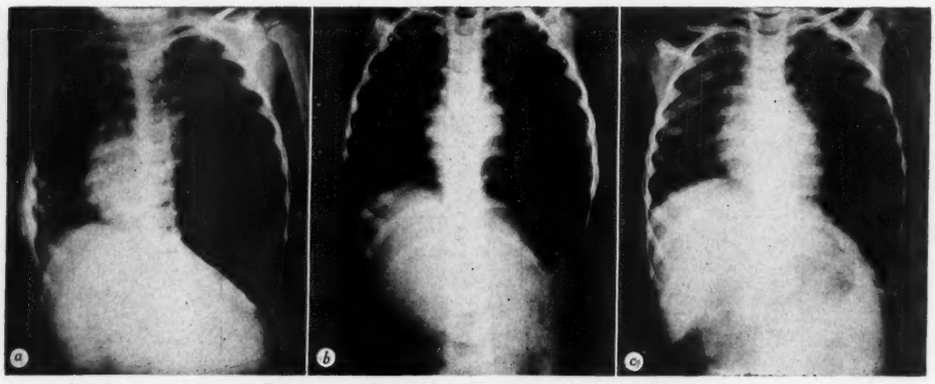

FIG. 41. Same case as that represented in Figure 40. *a*, roentgenogram taken immediately following operation (on operating table) showing complete collapse of left lung. Marked shift of heart and mediastinum to right. *b*, roentgenogram taken thirty minutes later than *a* after withdrawal of 700 cc. of air. Heart and mediastinum in normal position. Left lung still collapsed. *c*, roentgenogram taken three days after operation. Heart and mediastinum in midline. Partial expansion of the left lung.

there will be marked increase in the intra-abdominal pressure when the viscera are replaced into the abdomen.

In the smaller hernias of this type the opening can be closed without utilizing interruption of the phrenic nerve. In the larger hernias it is a necessary procedure. The opening is completely closed by overlapping the margins from 2 to 3 cm. In infants this closure is made with interrupted silk. Before the opening is completely closed, the air is aspirated from the pleural cavity by inserting a catheter connected to a suction apparatus. At the time of withdrawal of the catheter the last suture is tied, completely closing the communication between the thorax and the abdomen. (Fig. 41.)

One of the chief dangers associated with the repair of these hernias is marked alteration of intrathoracic or intra-abdominal pressure. It is very important in these cases that the respiratory function be maintained by positive pressure during the operation and that at the completion of the operation a negative pressure be obtained and secured in the thoracic cavity. A roentgenogram should be taken at the completion of the operation to see that there is no shift of the mediastinum due to the pneumothorax. I do not permit the patient to leave the operating table until I have seen the roent-

genogram. If there is any shift in the mediastinum, more air is withdrawn to maintain the mediastinum in the midline.

*Congenital Absence of the Posterior Half of the Diaphragm.* This

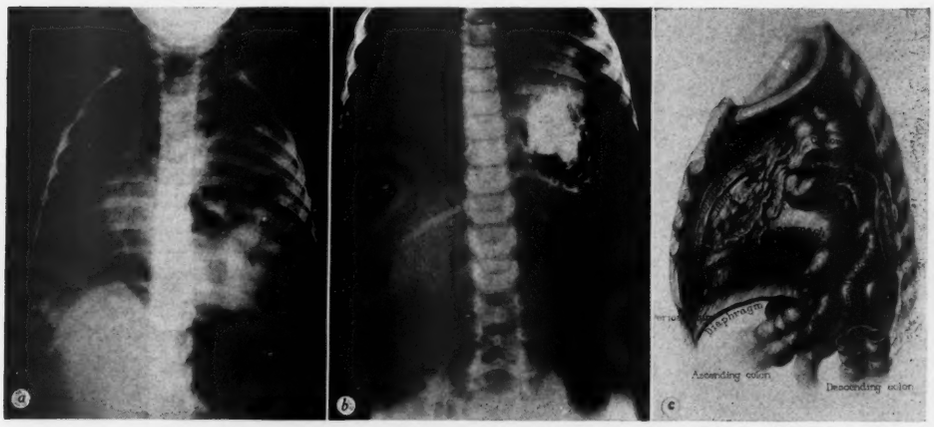


FIG. 42. *a*, patient aged 3 years. Congenital absence of portion of diaphragm. Left side of thoracic cavity almost completely filled with hollow viscera displacing the mediastinum to the right. *b*, same case. Entire stomach in left side of thoracic cavity. Colon distended with air and seen to the outer side and extending above the stomach. *c*, same case at operation. Eventration of colon, small bowel, stomach and spleen into left side of thoracic cavity, completely collapsing lung. Left kidney retropleural.

type of hernia is due to failure of the formation of that portion of the diaphragm which is derived from the pleuroperitoneal membrane. The defect is in the posterolateral portion of the diaphragm and usually extends from the eighth rib posteriorly and medially to the esophageal hiatus. These hernias usually do not have a sac but there may be an imperfectly developed enveloping membrane of peritoneum and omentum which simulates a sac. These hernias may be considered an enlargement of the foregoing pleuroperitoneal type in that the essential difference is a much more extensive congenital defect in the formation of the diaphragm. There are more abdominal viscera involved in the hernia in that these hernias always contain the stomach and spleen as well as the large and small bowel and occasionally the left kidney is elevated above its normal level into the pleural cavity. (Fig. 42.)

Not only do the surgical problems associated with these hernias involve all of the problems of the pleuroperitoneal hernias as far as altered intra-abdominal and intrathoracic pressure is concerned but in addition there is the problem of closing this large gap with the diaphragmatic muscle that is present and of reconstructing the attachment of the diaphragmatic muscle to the chest wall. (Fig. 43.)

In some instances the posterior perirenal fascia may be utilized in obtaining this closure and fixation to the chest wall. This can be accomplished if the gap is not too great by complete, permanent

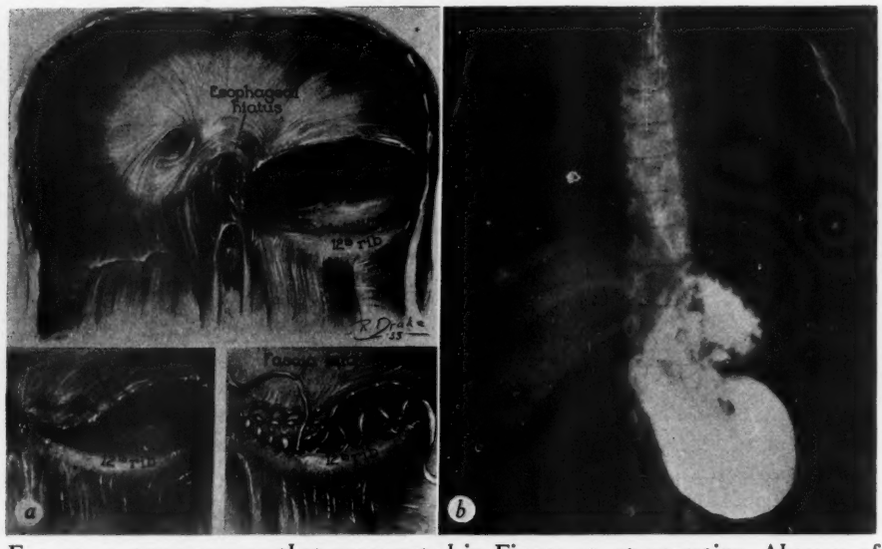


FIG. 43. *a*, same case as that represented in Figure 42, at operation. Absence of posterior part of left side of diaphragm. Repair of defect by suturing posterior margin of diaphragm to intercostal muscles with fascia lata. *b*, same case after operation. Stomach in normal position below the left side of diaphragm which is in normal relation to right side of diaphragm. Heart and mediastinum in normal position. Lung fields negative.

interruption of the phrenic nerve. If the defect is too large to permit the relaxed diaphragm to span this gap, it is necessary to shorten the diameter of the diaphragm by an extrapleural rib resection.

*Substernal Hernia through the Foramen of Morgagni (Larrey's Space).* This is one of the rarest types of diaphragmatic hernia. It is usually seen in adult life but is essentially congenital in origin. The opening in the diaphragm is usually immediately beneath the right side of the sternum and right costal margin through a slit in the anterior portion of the diaphragm (foramen of Morgagni, Larrey's space), or there is a linear separation of the diaphragm from the anterior chest wall extending entirely beneath the sternum more to the right side of the midline. These hernias always have a hernial sac of peritoneum and the round ligament of the liver is usually incorporated in the peritoneum which forms the sac.

The herniated abdominal viscera which are most commonly found in these hernias are the omentum and transverse colon. There may be nonrotation of the colon with herniation of cecum, appendix,

terminal parts of the ileum, ascending colon and part of the transverse colon. When the omentum only is contained in the hernia, the condition is not uncommonly thought to be an intrathoracic tumor.

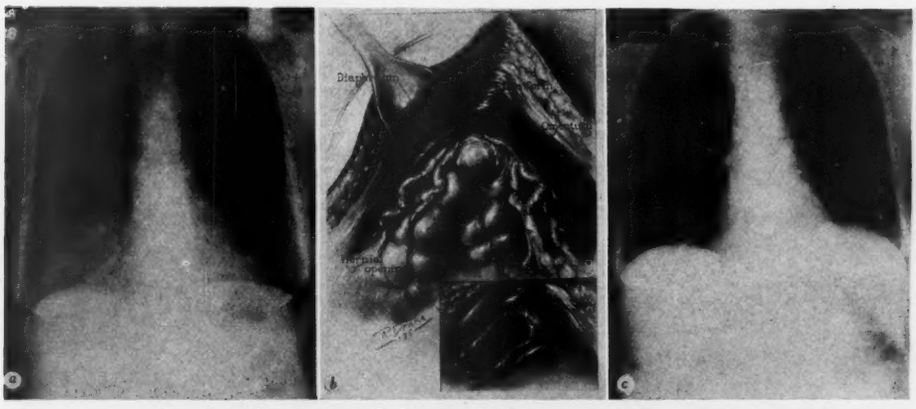

FIG. 44. *a*, patient aged *55* years. Substernal (foramen of Morgagni—Larrey's space) diaphragmatic hernia. Rounded shadow in right lower quadrant of thorax above diaphragm obscuring right border of heart and lung field. *b*, same case at operation. Abdominal approach. Herniation of omentum and colon through opening to right of ensiform cartilage. *c*, same case after operation. Right lung entirely expanded. Diaphragm, heart and lung fields negative.

In cases in which the colon is herniated, the condition can be recognized from the roentgenogram for the colon may be seen to extend to the top of the thoracic cavity on the right side. These hernias are repaired through right rectus incisions. The herniated omentum and colon are replaced into the abdomen. If the hernial sac is small, it can be inverted and excised. In the larger hernias in which the sac extends high into the pleural cavity, it is best to leave the sac in situ because of the danger of hemorrhage which may be associated with its removal or of injury to the pleura. In these cases the round ligament is cut and later sutured to the abdominal wall. The hernial opening in the diaphragm is closed with interrupted linen sutures. The closure is reinforced with a continuous layer of fascia lata removed from the thigh. The round ligament of the liver is then sutured over this closure. In cases in which there is a very large opening, it may be necessary to suture the overlapped margins of the opening to the chest wall in order to obliterate the hernial orifice completely. (Fig. 44.)

*Traumatic Diaphragmatic Hernia.* These hernias may result from direct or indirect injury to the diaphragm. The hernia may occur in either the right or the left side of the diaphragm but it

much more commonly occurs in the left side. The laceration may occur in any portion of the diaphragm but it is most common in the posterolateral portion and dome of the diaphragmatic muscle. The

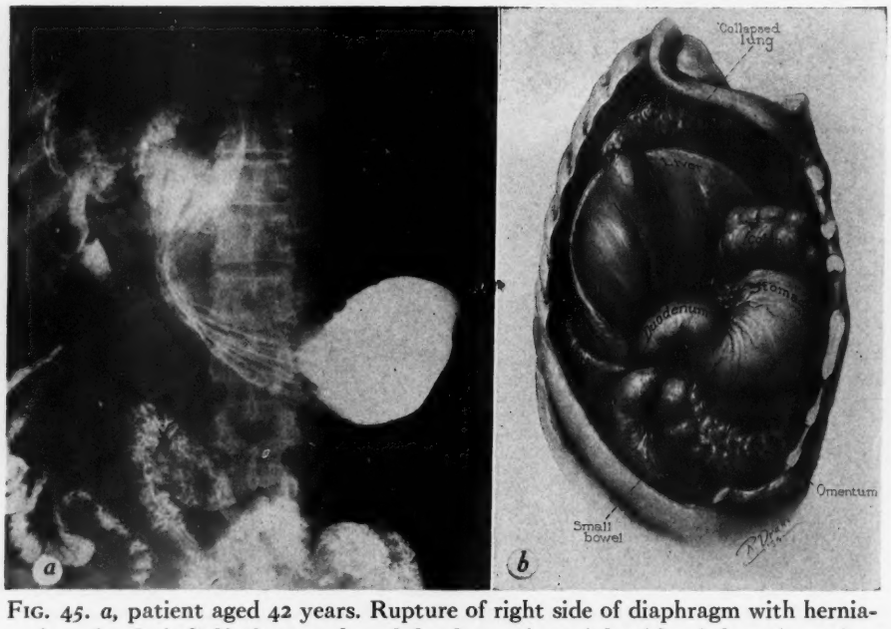


FIG. 45. *a*, patient aged 42 years. Rupture of right side of diaphragm with herniation of pyloric half of stomach and duodenum into right side of thoracic cavity. *b*, same case at operation. Liver, gallbladder, stomach, duodenum, pancreas and colon in right pleural cavity causing collapse of right lung.

laceration may extend across the entire leaf of the diaphragm and occasionally extends into the opposite side of the diaphragm. These lacerations occasionally split the esophageal hiatus but in most instances the circular muscle of the hiatus remains intact. The laceration involves the entire width of the diaphragmatic muscle producing a direct communication between the abdominal and the thoracic cavity and there is no hernial sac. The condition may be more properly termed an evisceration than a true hernia.

Any of the abdominal organs but those in the pelvis may be involved in these hernias. The most common are the stomach, large and small bowel, spleen and liver. The herniated abdominal viscera may entirely fill the hemithorax and extend to the apex of the thoracic cavity causing complete collapse of the lung with marked displacement of the mediastinum.

The surgical approach to these hernias may be through the thorax or through the abdomen. For all hernias through the right side of the diaphragm, I prefer the thoracic approach because the

large right lobe of the liver interferes with the exposure of the right side of the diaphragm. In hernias of this side the right lobe of the liver is often incorporated in the hernia and its reduction is more

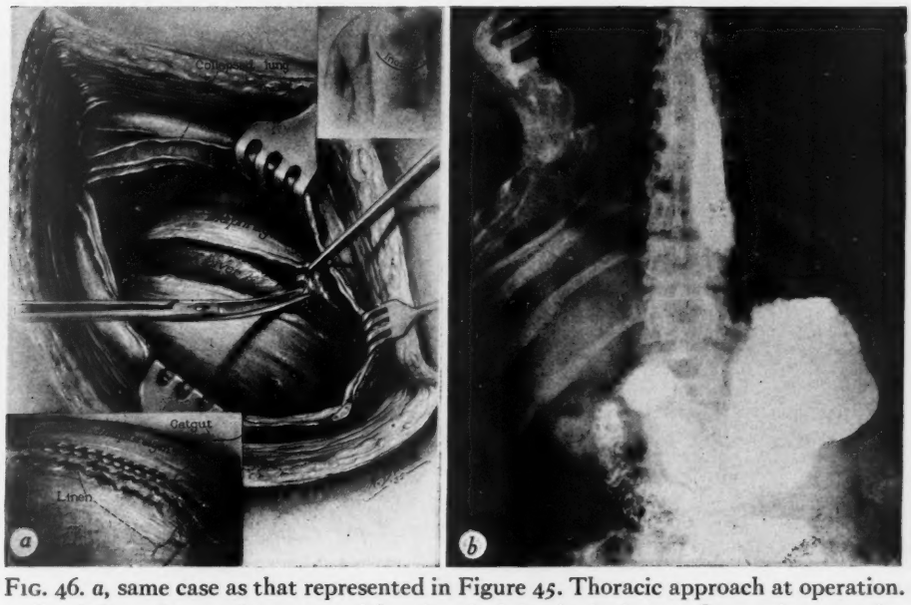


FIG. 46. *a*, same case as that represented in Figure 45. Thoracic approach at operation. Condition after replacement of herniated abdominal viscera. Separating margins of laceration from liver and overlapping repair of the diaphragm. *b*, same case after operation. Stomach and colon in normal position below the diaphragm. Partial expansion of right lung and thoracic deformity resulting from rib resection.

safely accomplished through the thoracic approach because there is less danger of hemorrhage from injury to the liver. (Figs. 45 and 46.)

In all traumatic hernias through the left side of the diaphragm, I prefer the abdominal approach through an oblique left rectus incision. The herniated viscera are usually very adherent to both the abdominal and the thoracic side of the diaphragm and to the structures within the thorax. The adhesions to the margins of the opening and to the under surface of the diaphragm are often very marked and should be separated first. The adhesions to the structures within the thoracic cavity are separated from below upward by approaching them through the hernial opening. By the abdominal approach this can be accomplished with little danger or injury to the abdominal or thoracic viscera, because the definite relationship of the herniated structures can be established.

In cases in which there has been considerable loss of structure or in which the muscle has been torn from its attachment to the

thoracic wall, the defect in the diaphragm should be repaired by fascia lata stabilized with linen sutures. I believe this to be the most satisfactory type of closure in all these cases. In cases of traumatic

FIG. 47. *a*, patient aged 44 years. Traumatic hernia through left side of dome of diaphragm with herniation of entire stomach into left side of thoracic cavity extending to third rib. *b*, same case. Lateral view of thorax revealing large loop of colon herniated into left side of thoracic cavity and extending to third rib. *c*, same case at operation. Abdominal approach. Left lobe of liver has been separated from diaphragm. Removal of adherent stomach and spleen from hernial opening.

hernia in which the laceration is confined to the dome of the diaphragmatic muscle, it usually is advisable to repair the opening by lapping the anterior margin over the posterior margin of the opening. When possible, it is advisable to overlap the margins of the opening from 2 to 3 cm. In those cases in which the laceration splits the muscle of the esophageal ring, great care should be taken in repairing the esophageal hiatus. In those cases in which the laceration extends to the margin of the thorax and in which the attachments of the diaphragm are torn from the thoracic wall, the repair is made not only by overlapping the laceration of the leaf of the diaphragm, but by resuturing the diaphragmatic muscle to the thoracic wall. This can be accomplished by suturing the diaphragmatic muscle (Figs. 47 and 48) to the intercostal muscles between the ribs. When possible, the diaphragmatic muscle should span two interspaces, being fixed to the intercostal muscles with fascia lata and stabilized with interrupted linen sutures.

In a few instances the relaxation of the diaphragmatic muscle caused by interruption of the phrenic nerve will not be sufficient for repair of the defect. In these cases the diameter of the thorax must be narrowed by resecting the lower ribs by thoracoplasty. It is usually not necessary to resect more than a few inches of the eighth, ninth and tenth ribs at the angles.

*Inflammatory Necrosis.* One of the rarest types of diaphragmatic hernia is that resulting from inflammatory necrosis of the diaphragm caused by the rupture of a subphrenic abscess on the

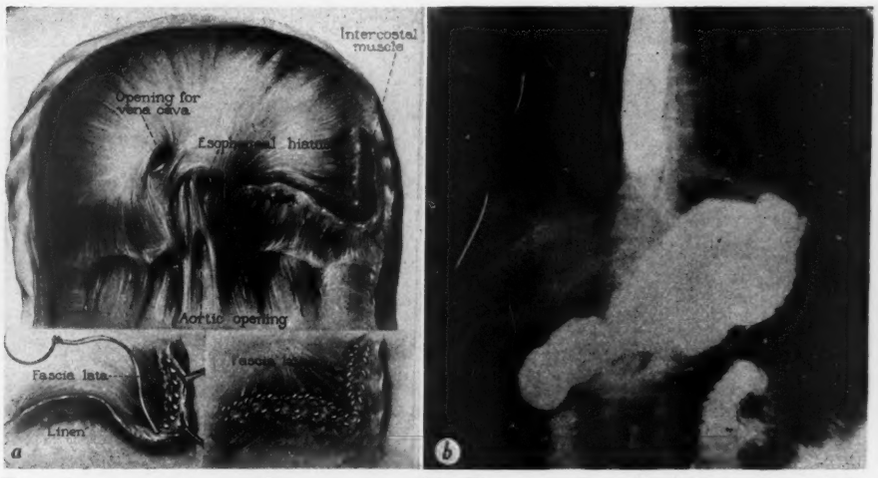


FIG. 48. *a*, same case as that represented in Figure 47. Repair of triangular rent in diaphragm with fascia lata by overlapping and suturing to chest wall after phrenicotomy. *b*, same case on dismissal. Entire stomach in normal position below the diaphragm. Pulmonary fields negative.

left side. This type of hernia may be termed a traumatic hernia. The acute abdominal condition may have preceded the diagnosis of hernia by months or years. These hernias often present a very difficult surgical problem because of the enormous number of adhesions which fix the herniated abdominal viscera to the under surface of the diaphragm as well as to the hernial orifice and at times to the under surface of the lung. These hernias usually occur in the dome of the diaphragm and do not involve the hiatus. They are repaired by overlapping the margins of the hernial orifice after carefully dissecting the viscera from the diaphragm and the margins of the opening. (Fig. 49.)

In all of these cases in which there has been a direct communication between the abdominal and the thoracic cavity, every effort should be made to reestablish the negative pressure within the pleural cavity by removing the air and by expanding the lung before the opening in the diaphragm is closed completely. In some instances this cannot be accomplished until after the rent in the diaphragm has been closed. In some cases pneumothorax may push the mediastinum and heart to the opposite side and cause marked embarrassment of respiration and circulation. In these cases it is

imperative that the mediastinum be stabilized in the midline immediately by aspirating the air from the pleural cavity with a needle until the pressure is negative. I think it advisable to make a

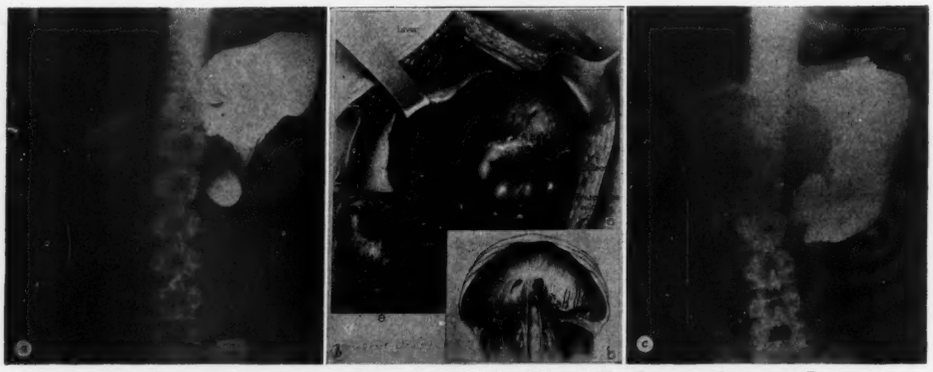

FIG. 49. *a*, patient aged 22 years. Rupture of left side of diaphragm due to inflammatory necrosis. Herniation of two-thirds of cardiac end of stomach, through opening in dome of left side of diaphragm, into left side of thoracic cavity. *b*, same case at operation. Abdominal approach. Stomach firmly adherent to margins of ruptured diaphragm. Opening situated posteriorly and repaired by overlapping closure. *c*, same case after operation. Stomach in normal position below repaired left side of diaphragm.

roentgenogram before the patient leaves the operating table, for the purpose of determining the amount of pulmonary expansion present.

Before closing the abdomen, the herniated viscera should be thoroughly explored, to be certain that there has been no injury to a viscus or that there are no bands of adhesions which will interfere with the function of the abdominal viscera. In cases in which there has been considerable obstruction of the large bowel, it may be necessary to perform appendicostomy or colostomy at the time of operation.

### POSTOPERATIVE MANAGEMENT

Most patients are given a blood transfusion either during or immediately following the operation. The blood of every patient is grouped for transfusion before the operation. If the systolic blood pressure decreases to 90 mm. of mercury or less, the patient should receive a transfusion of blood. I believe it is very important to maintain blood pressure at a fairly constant level and not permit it to drop more than 20 mm. of mercury below the normal preoperative reading. I prefer the use of blood transfusions to the use of other intravenous solutions or of drugs to maintain the blood pressure.

Blood transfusions are also advisable in all cases in which the hernia is associated with blood loss producing secondary anemia.

If there is excessive mucus in the bronchi and lungs at the time of operation, it is imperative that it be removed by aspiration through the intratracheal tube used for the administration of the anesthetic agent or by the use of the bronchoscope before the patient leaves the operating table.

On removing the patient to the hospital bed which has been made warm by hot water bottles, external heat is applied to the patient by use of additional hot water bottles. Fifty-eight to 60 per cent oxygen is administered in an oxygen tent until the patient has fully recovered consciousness and is continued thereafter as indicated. After the patient has fully regained consciousness, the oxygen may be administered with a nasal mask.

Care should be exercised in the selection and amount of sedation used. This is particularly important if there is any respiratory embarrassment. Sedation should be limited so as not to decrease the respiratory function. I prefer using codeine supplemented by a small amount of morphine to relieve severe pain. In all cases in which dilatation of the stomach was present at the time of operation, intermittent or continuous gastric lavage is employed to keep the stomach empty for the first three to five days after operation.

All patients are given glucose and physiologic salt solution intravenously for three to five days until they are able to take sufficient quantities of fluids by mouth. The giving of fluids usually is started within forty-eight hours after the operation. In cases in which there has been herniation into the thoracic cavity of a large portion of the abdominal viscera over a long period, the replacement of these viscera in the abdomen causes a marked increase in the intra-abdominal pressure, which may lead to partial or complete obstruction. In cases of partial obstruction, the condition may be relieved by conservative measures, but in cases of complete obstruction, it may be necessary to perform enterostomy in order to reduce the intra-abdominal pressure and to relieve the obstruction.

In all cases in which the herniated viscera are removed from the pleural cavity, and in some cases in which the herniated viscera are removed from the posterior portion of the mediastinum, such as would be the case in herniation through the esophageal hiatus, traumatic effusion occurs in the pleural cavity. The treatment of this effusion depends on the amount of respiratory embarrassment

associated with it. In most instances the effusion is slight; it will gradually become absorbed so that special treatment will not be required. In cases in which the effusion progresses to produce respiratory embarrassment, pleurocentesis, performed one or more times, is required. In some cases empyema may develop, requiring intercostal drainage, and possibly later rib resection. In my experience empyema has rarely occurred in cases in which the hernia was repaired by the abdominal approach.

In some cases atelectasis may be caused by the lodgment of mucus in a bronchus. In most of such cases the condition will respond to conservative treatment. It may sometimes be necessary to remove the mucus by bronchoscopic aspiration.

### REVIEW OF THE OPERATIVE PROCEDURES EMPLOYED AND THE RESULTS OF OPERATION IN 250 CASES IN WHICH TREATMENT WAS SURGICAL

The operative procedures employed in the 250 cases in this series were as follows:

In 225 cases the patients were treated by radical operation. The herniated abdominal viscera were replaced in the abdomen and the abnormal opening in the diaphragm was repaired. In 133 of these cases the diaphragm was either temporarily or permanently paralyzed preliminary to operative repair of the hernia. In two cases it was necessary to perform extrapleural thoracoplasty in addition to interruption of the phrenic nerve as a preliminary procedure to repair of the hernia. In 223 cases the abdominal approach was employed to repair the hernia; in the remaining two cases a combined thoracic and abdominal approach was employed.

In fifteen cases it was necessary to perform other operative procedures at the time of repair of the hernia. In three cases gastric resection (Pólya type) was done, in one case for gastric ulcer at the lesser curvature of the stomach and in two cases for carcinoma of the pyloric end of the stomach. In three cases posterior gastroenterostomy was performed, in one case for high gastric ulcer involving the lower end of the esophagus and in two cases for a large duodenal ulcer causing almost complete obstruction of the pyloric end of the stomach. In five cases splenectomy was performed. In all of these cases the spleen was firmly adherent to the margins of the opening and to the thoracic side of the diaphragm. Trauma associated with its removal from the hernial orifice and the diaphragm necessitated the removal of the spleen in three cases and in two cases the spleen

was removed because of tuberculosis. In one case appendectomy was performed for subacute appendicitis and in one case appendicostomy was performed at the time of operation because of obstruction and marked dilatation of the colon.

In eleven cases moderate shortening of the esophagus was associated with the hernia. In ten of these cases the diaphragm could be sutured entirely above the stomach after the diaphragmatic muscle had been paralyzed by interruption of the phrenic nerve. In one case a small portion of the cardia was incorporated in the closure of the hernial orifice.

TABLE II

SURGICAL PROCEDURES AND OPERATIVE RESULTS IN 250 CASES

| | | |
|---|---|---|
| Radical repair of defect in diaphragm | | 225 |
| Approach: Abdominal, 223; thoracic, 2 | | |
| Preliminary interruption of phrenic nerve | 133 | |
| Preliminary extrapleural thoracoplasty | 2 | |
| Operations in conjunction with repair of hernia: | | |
| Gastric resection for gastric ulcer (1) for carcinoma (2) | 3 | |
| Closure of perforated gastric erosion (2) (total erosions, 23) | 2 | |
| Gastroenterostomy for gastric ulcer (1) for duodenal ulcer (2) | 3 | |
| Splenectomy for tuberculosis (2) for injury (3) | 5 | |
| Appendicostomy for obstruction | 1 | |
| Appendectomy for appendicitis | 1 | |
| Interruption of left phrenic nerve (hiatus hernia); palliative, 7; therapeutic, 18 | | 25 |
| Total patients subjected to operation | | 250 |
| Recurrence of hernia according to type subjected to operation: | | |
| Traumatic hernia, 0; congenital defect, 0; esophageal hiatus, 7 | | |
| Recurring esophageal hiatus hernias (7) | | |
| (Roentgenologic diagnosis without recurrence of symptoms, 4) | | |
| (Roentgenologic diagnosis with recurrence of symptoms, 3) | | |
| Repair of recurrent hernias, 2 | | |
| Operative deaths, 10 or 4 per cent | | |

Twenty-five patients with esophageal hiatus types of hernia were treated conservatively. In these cases interruption of the left phrenic nerve was done as a palliative or therapeutic measure; in seven of these cases, it was the only procedure contemplated, as radical operation was contraindicated, and in the remaining eighteen cases the procedure was in the nature of a therapeutic test. It may be necessary to perform radical repair of the hernia in some of these cases later to obtain complete relief of symptoms.

There were ten deaths, or a mortality rate of 4 per cent, following operation; seven deaths occurred in cases of esophageal hiatus hernia, two in cases of traumatic hernia and one in a case of hernia

through the pleuroperitoneal hiatus. Nine of the deaths were due to pulmonary congestion or pneumonia with respiratory and cardiac failure; one of these patients had cardiac disease and one had cardiorenal disease prior to operation. One death was due to cerebral embolism. (Table II.)

Of the 215 cases in which patients recovered from radical repair of the hernia, a small recurrence has developed subsequently in seven of the esophageal hiatus type. These recurrences occurred from three months to five years after operation. Of these seven cases of recurrence, in four cases the recurrence was small and it was recognizable by roentgenographic examination only as there was no definite recurrence of the former symptoms to indicate its presence. In three cases there was a moderately large recurrence of the hernia discovered by roentgenologic examination and definite recurrence of the patient's symptoms. In two of these cases the recurrence followed an attack of an influenzal type of pneumonia which occurred three and five months, respectively, after operation. The pneumonia was accompanied by severe attacks of coughing which could account for the recurrence. In these two cases the symptoms became severe enough to require a second repair of the hernia, which has been done. Following the second operation there has been no further recurrence clinically or roentgenologically. In the third case there is no known cause for the recurrence of the hernia. The patient continues to have moderately severe symptoms and will probably ultimately require a second repair of the hernia.

There were no recurrences of the hernias in any of the traumatic or congenital types of hernia due to structural deficiencies of the diaphragm.

Of the 215 patients who recovered from radical repair of all of the different types of diaphragmatic hernia, 212 were relieved of symptoms. All patients have been examined roentgenologically from six months to a year after operation and at periodic intervals since that time.

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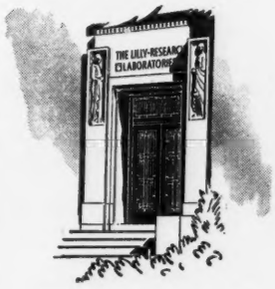


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# SOCIETY MEETINGS

AMERICAN ACADEMY OF ORTHOPAEDIC SURGEONS
*Boston*, January 21–25, 1940. (Secy.—Dr. Carl Badgley, University Hospital, Ann Arbor, Mich.)

AMERICAN ASSOCIATION FOR THE STUDY OF GOITER
*Rochester, Minn.*, April 15–17, 1940. (Secy.—Dr. W. Blair Mosser, Kane, Penna.)

AMERICAN ASSOCIATION FOR THE SURGERY OF TRAUMA
*Atlantic City*, June 7–8, 1940. (Secy.—Dr. Ralph G. Carothers, 409 Broadway, Cincinnati)

AMERICAN ASSOCIATION OF GENITO-URINARY SURGEONS
*Skytop, Penna.*, June 20–22, 1940. (Secy.—Dr. Charles C. Higgins, 2020 East 93rd Street, Cleveland)

AMERICAN ASSOCIATION OF INDUSTRIAL PHYSICIANS AND SURGEONS
*New York*, June 4–7, 1940. (Secy.—Armour G. Park, 540 N. Michigan Ave. Chicago)

AMERICAN ASSOCIATION OF OBSTETRICIANS, GYNECOLOGISTS AND ABDOMINAL SURGEONS
*Excelsior Springs, Mo.*, September 19–21, 1940. (Secy.—Dr. James R. Bloss, 418—11th St., Huntington, W. Va.

AMERICAN ASSOCIATION OF RAILWAY SURGEONS
*Chicago*, September 16–18, 1940. (Secy.—Dr. D. B. Moss, 547 W. Jackson Blvd., Chicago)

AMERICAN COLLEGE OF SURGEONS
*Chicago*, October 21–25, 1940. (Secy.—Dr. Frederic A. Besley, 215 N. Sheridan Rd., Waukegan, Ill.)

AMERICAN CONGRESS OF PHYSICAL THERAPY
*Cleveland*, September 2–6, 1940. (Secy.—Dr. Richard Kovacs, 2 East 88th Street, New York)

AMERICAN GYNECOLOGICAL SOCIETY
*New York*, June 17–19, 1940. (Secy.—Dr. Richard W. TeLinde, 11 East Chase St., Baltimore)

AMERICAN MEDICAL ASSOCIATION
*New York*, June 10–14, 1940. (Secy.—Dr. Olin West, 535 N. Dearborn Street, Chicago)

AMERICAN ORTHOPAEDIC ASSOCIATION
*Kansas City*, May 6–9, 1940. (Secy.—Dr. Ralph K. Ghormley, The Mayo Clinic, Rochester, Minn.)

AMERICAN PROCTOLOGIC SOCIETY
*Richmond*, June 9–11, 1940. (Secy.—Dr. Curtice Rosser, 710 Medical Arts Bldg., Dallas)

THE AMERICAN SOCIETY OF ANESTHETISTS, Inc.
*New York*, December 12, 1940. (Secy.—Dr. Paul M. Wood, 745 Fifth Avenue, New York)

AMERICAN SURGICAL ASSOCIATION
*St. Louis*, May 1–3, 1940. (Secy.—Dr. Charles G. Mixter, 319 Longwood Avenue, Boston)

ASSOCIATION OF MILITARY SURGEONS OF THE U. S.
*Cleveland*, October, 10–12, 1940. (Secy.—Dr. H. L. Gilchrist, Army Medical Museum, Washington, D. C.)

CONGRESS OF ANESTHETISTS
*Chicago*, October 21–25, 1940. (Secy.—Dr. E. Klaus, 1699 W. 25th Street, Cleveland)

INTERNATIONAL COLLEGE OF SURGEONS
*Venice, Fla.*, February 11–14, 1940. (Secy.—Dr. Charles H. Arnold, 903–15 Terminal Bldg., Lincoln, Nebr.)

INTER-STATE POST-GRADUATE MEDICAL ASSOCIATION OF NORTH AMERICA
*Cleveland*, October 14–18, 1940. (Secy.—Dr. Tom B. Throckmorton, 406 Sixth Avenue, Des Moines)

NEW ENGLAND SURGICAL SOCIETY
*Poland Spring, Me.*, September 27–28, 1940. (Secy.—Dr. John F. Gile, Hanover, New Hampshire)

PACIFIC COAST SOCIETY OF OBSTETRICS AND GYNECOLOGY
*San Francisco*, November 6–9, 1940. (Secy.—Dr. T. Floyd Bell, 400—29th Street, Oakland, Calif.)

PACIFIC COAST SURGICAL ASSOCIATION
*Portland*, April 3–6, 1940. (Secy.—Dr. H. Glenn Bell, University of California Hospital, San Francisco)

PAN-PACIFIC SURGICAL ASSOCIATION
*Honolulu*, 1942. (Secy.—Dr. Forrest J. Pinkerton, Young Bldg., Honolulu)

SOUTHEASTERN SURGICAL CONGRESS
*Birmingham*, March 11–13, 1940. (Secy.—Dr. B. T. Beasley, 701 Hurt Bldg., Atlanta)

SOUTHERN SURGICAL ASSOCIATION
*Hot Springs, Va.*, December 10–12, 1940. (Secy.—Dr. Alton Ochsner, 1430 Tulane Avenue, New Orleans

WESTERN SURGICAL ASSOCIATION
*Topeka, Kans.*, December 6–7, 1940. (Secy.—Dr. Albert H. Montgomery, 122 S. Michigan Blvd., Chicago)

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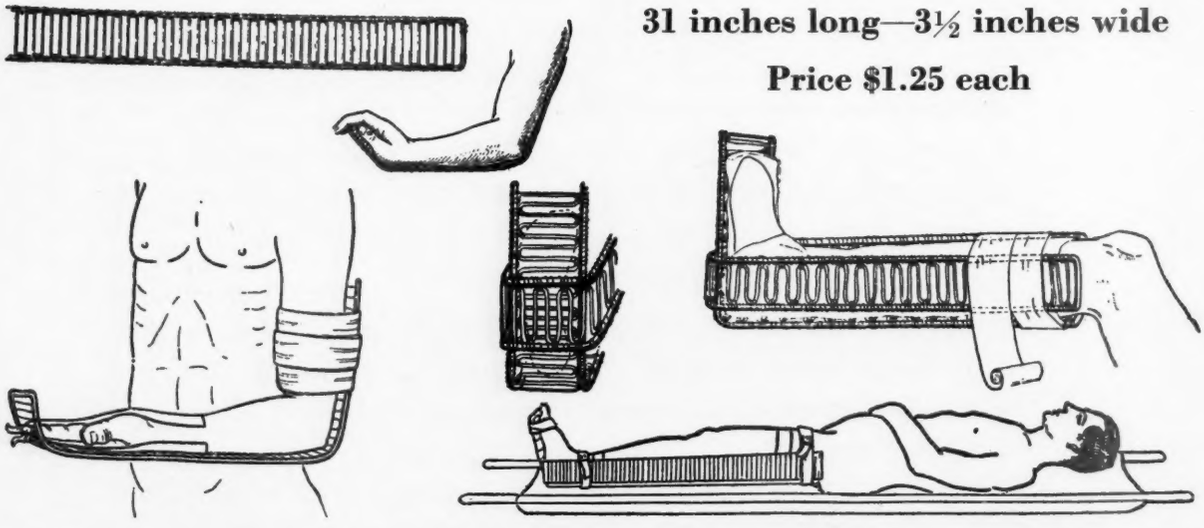

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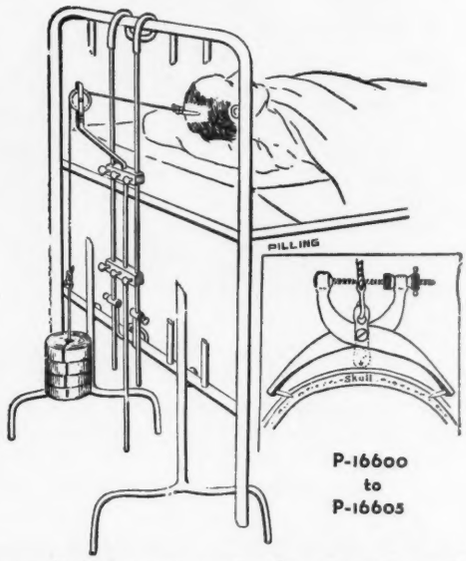


P-16600
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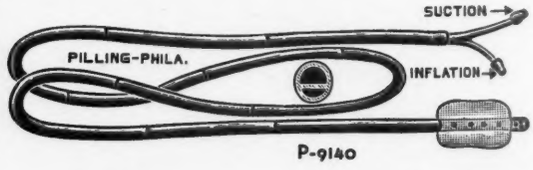


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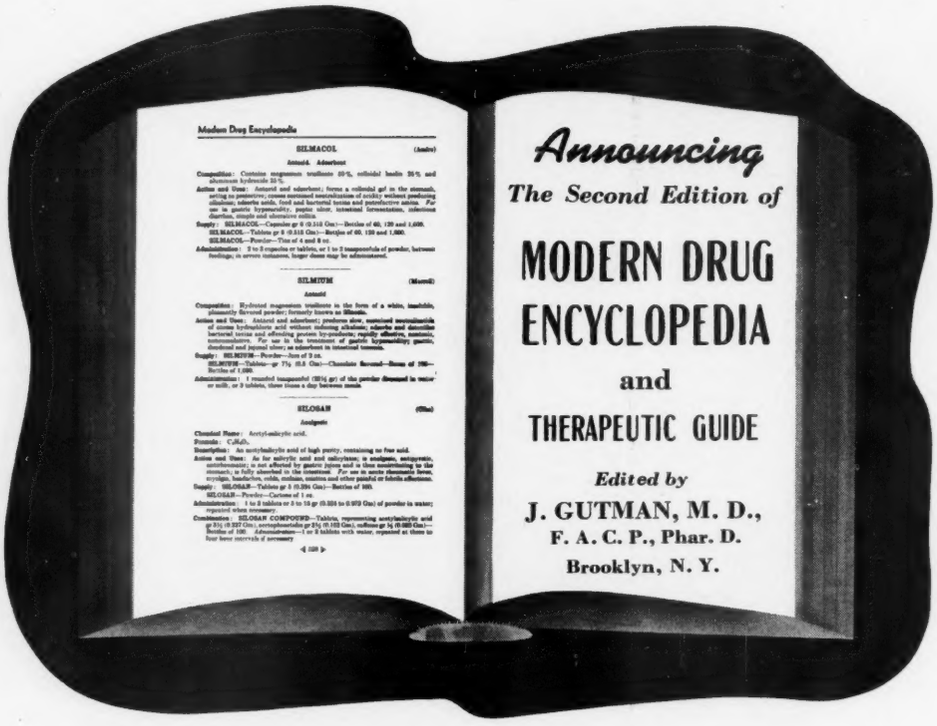


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*Campbell, W. C. and Speed, J. S., Annals of Surgery, 110-1. July, 1939. • Berry, H. C., Journal of Arkansas State Dental Association, 10-2. June, 1939. • Strock, A. E., American Journal of Orthodontics & Oral Surgery, 25-5. May, 1939. • Doherty, W. B., American Journal of Ophthalmology, 22-4. April, 1939. • Smith-Petersen, M. N., The Journal of Bone & Joint Surgery, 21-2, April, 1939. • Claflin, R. S., American Journal of Orthodontics & Oral Surgery, 25-1. January, 1939. • Hopkins, H. H. and Zuck, F. N., Medical Bulletin of Veterans' Administration, 15-3. January, 1939. • Venable, C. S. and Stuck, W. G., Journal of American Medical Association, 11-15. October 8, 1938.

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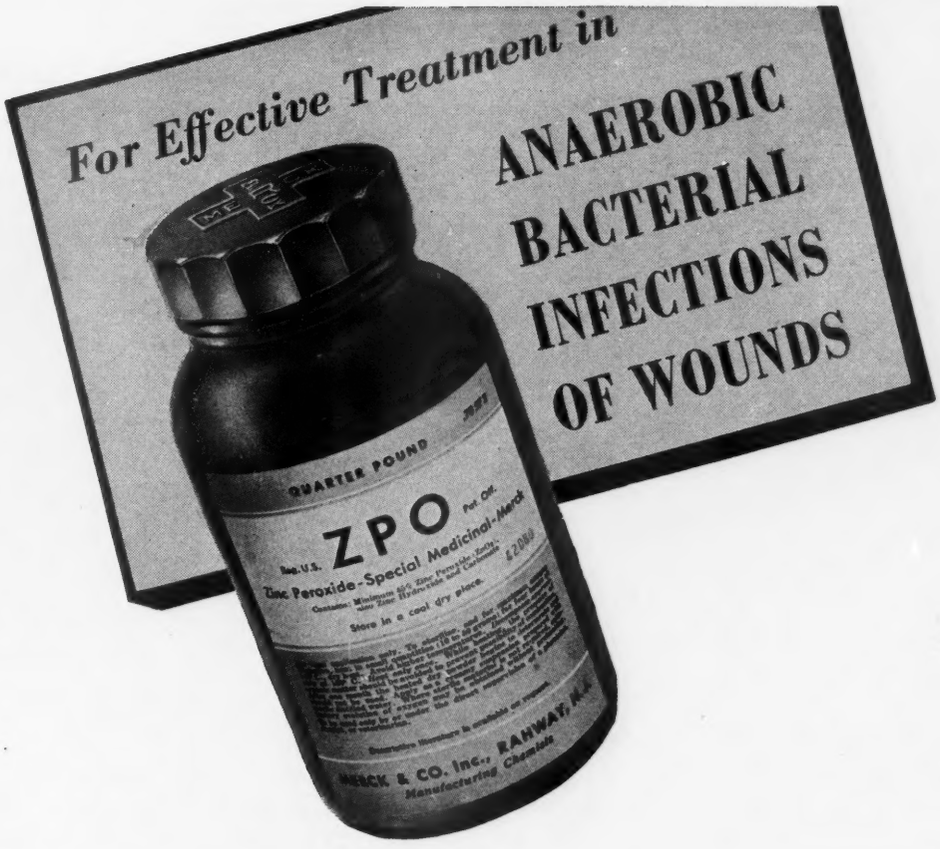


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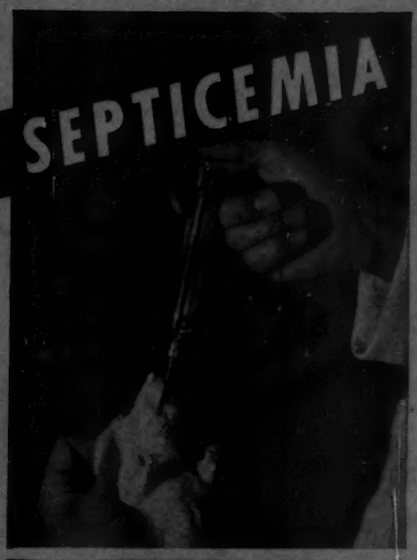

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*Spink, W. W., and Hansen, A. E.: Sulfathiazole, Clinical Evaluation. *J.A.M.A.*, 115:840, Sept. 7, 1940.

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